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A PLEA FOR A MORE INTENSIVE STUDY OF SURGICAL PATHOLOGY.[1]

By H. S. STACY, M.D., Ch.M. (Sydney), F.R.A.C.S.,

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WHILST thoroughly agreeing with Moynihan that the study of the pathology of the living is of immense importance and has in bygone years not received the attention it deserved, yet I feel that pathology as revealed in the autopsy room is also neglected to a much greater extent than it should be. As contrasted with that of the living, it has the disadvantages: (i) that few of the earlier changes are seen, (ii) that the finer alterations of colour are lost, (iii) that catarrhal changes often cannot be recognized. On the other hand, pathological changes on the dead body are capable of being demonstrated in the *post mortem* room to a much larger number than in the operating theatre, and a degree of examination and manipulation is possible that, of course, is far beyond what is possible in the living body.

By correct interpretation of our modern tests (radiological, hæmatological, electrical, biochemical *et cetera*) we attempt to visualize the state and function of the various organs, but with what limited success in the case of some of them, most of us are only too painfully aware.

Can we do anything to improve our knowledge in a branch of surgery in which the average Australian surgeon does not compare so favourably with the British surgeon as he does in his operative dexterity. This is the universal opinion of all who have travelled. Some may have a flair for surgical diagnosis and judgement, gained from a long and varied experience, but to most it must come through a

[1] Read at a meeting of the Queensland Fellows of the Royal Australasian College of Surgeons, August 17, 1934.

sound knowledge of pathology coordinated with clinical experience. Is it not a fact that material is going out of the back gates of our hospitals every day without that complete *post mortem* examination from which invaluable lessons might be learned, especially if coordinated with a close study of the clinical history? Without this latter the whole value is not obtained. We may never obtain a *post mortem* examination in all cases, but we should make better use of those we get.

We have an abundance of laboratory tests which we perform on the living body, the value of some of which is doubtful. Viewed in the cold, hard light of the autopsy room, some of them are not impressive in their information. I may be wrong, but my own feeling is that our tests of liver function are not very helpful; there seems to be scope for a lot of work to be done in this direction.

Where renal function used to be largely guess work and clinical acumen, the tests now are tremendously helpful, though even these (or at any rate their interpretation) are occasionally not in conformity with the *post mortem* examination.

I will make a few brief general remarks about the various organs and some of their functional tests.

Heart.

The diagnosis of cardiac failure is not uncommon on surgical death certificates; there is at times a doubt whether this was not really due to some operative complication, such as hæmorrhage *et cetera*.

The truth is that, although the capacity of the patient to stand the operation often depends upon the state of the heart muscle, our means of estimating it are not very accurate; the electrocardiograph has undoubtedly added greatly to our knowledge. The difference between an examination such as a patient conceives is sufficient and the examination that is really necessary is very great. More information could be got, of course, from the patient by a carefully taken history, but this aspect of a case is too often neglected.

Of recent years coronary thrombosis has become very fashionable, but a visit to the *post mortem* room will show that though death may have been due to cardiac disease, coronary thrombosis is not so common as some think. The outstanding sign of its presence is the resultant infarct (coronary arteries being end arteries).

Pancreas.

In diabetes it is not uncommon at an autopsy to find no macroscopic or microscopic evidence of any lesion (Figure I). Fibrosis is usually the result of pancreatitis and is not usually present in diabetes. It is by no means universal to find microscopic evidence of involvement of the islets of Langerhans (Figure II) in diabetes (Dr. Inglis is my authority for this statement). The main pathology of this disease that is evident is extensive endarteritis; where calcification is added to this, as is quite common, the X rays will divulge it, especially in the vessels of the limbs (Figure III). Presumably the endarteritis of the *vasa vasorum* and the consequent ischæmia lead to the deposition of calcium.

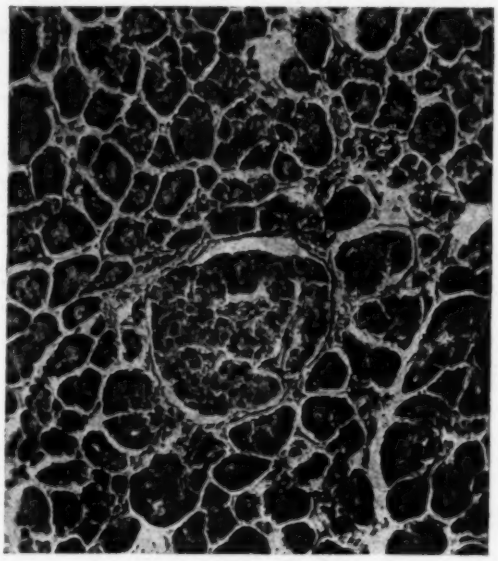

Figure I.
Section of normal pancreas, showing islet of Langerhans.

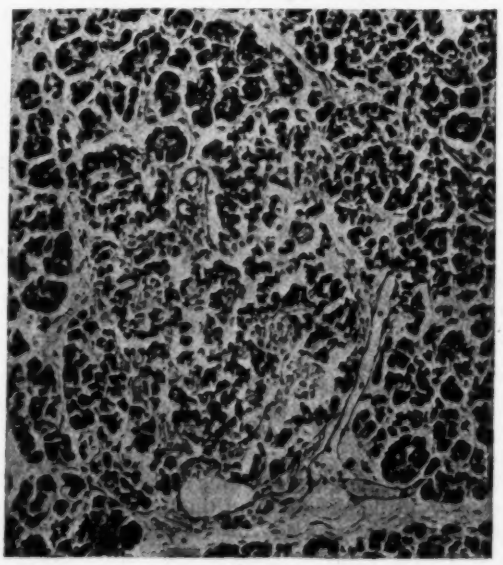

Figure II.
Pancreas, showing pathological islet of Langerhans in a case of diabetes.

Quite a reliable test for pancreatic disease is the examination of the fæces:

1. Rate of drying, which in the presence of an excessive amount of fat is prolonged beyond the normal two days.

2. Estimation of the percentage of total fat, which normally is in the vicinity of 25%; any marked increase is suggestive.

3. Estimation of the percentage of the fat which is unsplit; normally this is in the vicinity of 25%; in pancreatic disease there is usually a marked increase.

The diastatic index is raised only when the lesion has an obstructive effect upon the pancreatic duct. It is measured in the urine.

Liver.

Tests of liver function are not in common use, so presumably are not of great value.

Radiographic tests as to enlargement, especially in the effect that it has of pushing the dome of the diaphragm up, are very helpful. Sometimes, when a thoracic shadow runs into the liver shadow, it may be difficult to say whether the cause is supra- or infra-diaphragmatic (Figure IV). I saw this neatly solved in a recent case by the injection of air into the abdominal cavity; a subsequent radiograph showed a distinct curvilinear interval between the diaphragm and liver; the lesion was at the base of the lung (Figure V).

Kidneys.

Probably two of the most valuable tests of a simple nature are the phthalein and indigo-carmine tests; they need no laboratory for their performance.

The urea concentration, blood urea and creatinin estimations are very valuable; but I have seen the blood urea test let the surgeon down badly.

Scheme of Investigation.

My scheme is that, if possible, the surgeon should be present at the *post mortem* examination so as to see the organs fresh and *in situ*; if this is not possible, then they could be preserved for him in the refrigerator for some hours; after that the most important organs would be put in preservative.

A death meeting could be held, say, once a fortnight in the late afternoon. Prior to the meeting the surgeon in attendance would have had the opportunity of studying the clinical aspect of the case in view of the *post mortem* findings. There would be no discussion of the cases; those present would pass on from one to another.

I would analyse the cases under six headings, as follows:

1. Short clinical history read by the attending medical officer in charge of the case.

2. Account of the autopsy by the pathologist.

3. State of the organs as expected by the attending medical officer.

4. Data on which these beliefs were founded (medical examination, pathological and other tests).

5. Comments as to the help (or otherwise) of the tests in this particular case.

6. Clinical comments: (*a*) General. (*b*) Could a different procedure have saved life?

Illustrative Cases.

I shall now proceed to illustrate this plan by concrete cases and shall use these six headings in each instance.

Empyema.

1. *Short Clinical History.*—A.W., aged thirty-one years, an electrician, was admitted to hospital on June 2, 1934. He died on July 14, 1934. He had a sudden onset of pain on the right side of the abdomen one month before admission. Operation thirty-six hours later disclosed a ruptured appendix. A week later he developed severe pain in the right side of the chest; three weeks later he coughed up a large amount of purulent sputum, which had continued.

Physical examination revealed then a pale man with obvious respiratory distress. Physical signs of lung abscess were present at the right base. On July 6, 1934, one ounce of pus was aspirated from the right pleura. The sputum contained no tubercle bacilli. Pus from the pleura was sterile on July 7, 1934; it gave a pure culture of *Staphylococcus aureus* on July 14, 1934. Blood culture was sterile. The leucocytes numbered 32,880 per cubic millimetre. X rays revealed hydropneumothorax, also extensive mottling of both lungs, which was thought to be tuberculous.

2. *Account of the Autopsy.*—In the right pleural cavity, in the anterior portion of the lower third, there was a large loculated cavity containing over a pint of foul pus; the walls of the cavity were thick and were formed by the base of the lung, portion of the dome of the diaphragm and chest wall. The right lung was adherent to the pericardium. Both lungs showed marked anthracosis. The pericardium contained about 200 cubic centimetres of foul pus under increased pressure; the visceral pericardium was shaggy. The coronary arteries, valves and aorta were healthy. In the abdomen no pus was found; some adhesions of the small bowel were found in the pelvis.

3. *State of the Organs as Expected by the Attending Medical Officer.*—There was thought to be an abscess of the right lung; an empyema containing gas was recognized in the later stages. The lungs were strongly suspected of being tuberculous. No lesion of the pericardium was suspected.

4. *Data on which these Beliefs were Founded.*—The patient was thought to have a lung abscess because he expectorated pus (in reality it was an empyema communicating with a bronchus). The condition was thought to be tuberculous because of the extensive mottling shown by the X rays.

5. *Comments as to the Help (or otherwise) of the Tests in this Particular Case.*—The sputum (examined fifteen times) was found not to contain tubercle bacilli; this was correct. The radiographer thought the mottling was tuberculous; it was really due to anthracosis. The right-sided pleural opacity blurred the pericardial shadow, which in these cases of pericardial effusion is most marked at the base (that is, superiorly) and is somewhat rounded.

6. *Clinical Comments.*—(*a*) General. Close questioning would have revealed that the patient lived until lately near Glasgow, explaining the mottling of the lungs.

(*b*) Could a different procedure have saved life? As to the empyema, yes. Closed drainage with aspiration and the use of Dakin's solution might have been used; many might prefer closed drainage alone. As to the pericardial purulent effusion, I have never drained one, but have read of its being done successfully by removing the sternal end of the fifth, sixth and seventh costal cartilages. I used this approach successfully on one occasion for the removal of a bullet from the posterior wall of the pericardium, exposing the pericardial cavity.

Empyema with Cerebral Softening due to Infective Thrombosis.

1. *Short Clinical History.*—C. McC., aged seventy-three years, was admitted to hospital on May 16, 1934. He died on June 27, 1934. He complained of a cough for one month. He had a rigor and stabbing pain in the epigastrium the day before admission to hospital.

Physical examination revealed a right pleural effusion, with a swinging temperature; a subtympanitic note was present in the region of the inferior angle of the scapula. Some fluid was withdrawn (it proved to be sterile); the cells were principally neutrophile. As the temperature was still swinging five days later, the chest was aspirated again, 720 cubic centimetres (twenty-four ounces) of foul pus being withdrawn. A drainage tube was inserted two days later and open drainage was instituted. Much pus continued to be discharged. His general condition deteriorated.

Two and a half weeks after the operation he had a secondary hæmorrhage; this continued for four days, together with pus. Two weeks later he was found sitting propped up, unconscious and with a rapid, feeble pulse. Respiration was stertorous. For two weeks prior to the onset of coma his temperature had been approximately normal; with the bad turn it rose to 38·9° C. (102° F.). He died the following day.

X ray examination revealed complete opacity of the lower portion of the right lung up to the sixth rib in the mid-axillary line. There was slight deviation of the mediastinum to the left.

Examination of fluid from the chest on the second aspiration revealed *Staphylococcus aureus*, a streptococcus and *Bacillus coli communis*. The sputum contained no tubercle bacilli.

2. *Account of the Autopsy.*—Examination of the chest showed that the right lung was adherent at its base to the diaphragm; it was dark and gangrenous in places near the base; pus could be squeezed out of adjoining areas. On the posterior wall of the chest, separated off from the general pleural cavity, was a cavity, somewhat elongated, lying over the wound on the chest wall, but not drained by it (as it had partially closed); this contained about 60 cubic centimetres (two ounces) of foul blood-stained fluid. Examination of the heart revealed an old adherent pericardium; the heart muscle looked brownish; there were a few atheromatous patches in the coronary arteries; the aorta was healthy. In the brain there was softening of a considerable area of the white substance of the right cerebral hemisphere; also softening of some patches of the grey matter (reddish with punctate hæmorrhages). The basal arteries were not atheromatous. The kidneys shows some interstitial changes.

3. *State of the Organs as expected by the Attending Medical Officer.*—In the right lung he expected to find some congestion and compression of the lower lobe. He did not expect to find pus in the pleura. In the kidneys some interstitial changes were expected. In the brain he did not expect to find any lesion.

4. *Data on which these Beliefs were Founded.*—As the temperature had been normal for about two weeks and the sinus had ceased discharging it was assumed that the empyema had healed. He did not suspect any brain lesion complicating empyema from which the patient would die in twenty-seven hours. He was aware of the liability to get cerebral complications in cases of chest infections, but they are more usually of the nature of abscess and are more chronic in course.

5. *Comments as to the Help (or otherwise) of the Tests in this Particular Case.*—The radiological and pathological tests were very helpful. No tests for renal function were performed; if done, these would probably have revealed some hypofunction. A later X ray examination of the chest would probably have indicated that there was still a collection of pus in the pleura.

6. *Clinical Comments.*—(*a*) General. (*b*) Could a different procedure have saved life? If it had been recognized that the empyema was not cured, then treatment of it (say by combined closed drainage with Dakin's solution and aspiration or other approved treatment) would have cured the empyema and thus have saved the cerebral thrombosis, which was infective and due to a septic embolus coming from the chest cavity. I do not like open drainage; it favours mixed infection and delays the expansion of the lung.

Epithelioma of the Tongue, showing also Leucoplakia and Papillomata.

1. *Short Clinical History.*—J.B., aged sixty-seven years, was admitted to hospital on March 31, 1934. He died on May 21, 1934. He had had a thickening on the surface of the tongue for two years, also an ulcer on the left side of the tongue for seven weeks and a lump in the neck for seven weeks. He gave a history of gonorrhœa and syphilis and of urethral stricture. He was once a heavy smoker; latterly he had given it up. History of *potus* was slight. He had been a miner.

On examination the heart sounds were normal. The blood pressure was normal. Extensive leucoplakia was present on the dorsum of the tongue; several papillomata were also present, and an epithelioma on the left border. He had no teeth. In the neck the glands of the left digastric triangle and also the jugulo-digastric glands were enlarged and hard. In the chest there were signs of emphysema.

Electrocardiographic examination revealed left-sided preponderance and myocardial degeneration. The sputum contained no tubercle bacilli; in the culture the predominating organisms were staphylococci and streptococci. The blood gave no reaction to the Wassermann and Kahn tests. X ray examination of the chest revealed bilateral silicosis with enlarged bronchial glands.

On April 3, 1934, the glands of the neck were dissected out, the epithelioma of the tongue being excised a week later, the tongue wound being painted twice daily with gentian violet solution; in three weeks it was healed. He later developed pleuritic pain and signs of effusion on the left side. At this time the X ray report on the chest was: "Some consolidation of the left base; probably small amount of fluid; evidence of pulmonary tuberculosis at right apex." Nine days later 900 cubic centimetres (one and a half pints) of yellow turbid fluid were removed from the left pleural cavity by Potain's aspirator; the fluid contained staphylococci. The leucocyte count was 46,000 per cubic millimetre, of which 95% were neutrophile cells. The patient gradually sank and died.

2. *Account of the Autopsy.*—A fungating carcinoma was present on the left side of the neck. In the mediastinum the glands were enlarged and hard; they were invaded with epithelial newgrowth. The left pleura was filled with thick pus; the pleura was very much thickened and adherent to the diaphragm, chest wall and lung. The right side was normal. Both lungs showed septic pneumonia, also anthracosis; the right also had some bronchiectasis and invasion by squamous epithelioma. The left lung was also invaded by newgrowth. The heart muscle was flabby, atheroma was present on the aorta and valves. The coronary arteries were normal. The abdomen was normal. The liver showed chronic venous congestion. The kidneys were both congested; a slight increase of pelvic fat was present. The stomach and intestines were normal. The epigastric glands were enlarged and hard; they were not invaded.

3. *State of the Organs as expected by the Attending Medical Officer.*—In the left pleura he expected to find pus. In the lungs he expected to find signs of anthracosis and old tuberculosis at the right apex. In the heart some hypertrophy of the left ventricle was expected, with some dilatation of the right side.

4. *Data on which these Beliefs were Founded.*—Pus had been already withdrawn from the left pleura; it was expected to reaccumulate. The X ray examination of the lungs and the electrocardiographic report gave rise to the beliefs set out.

5. *Comments as to the Help (or otherwise) of the Tests in this Particular Case.*—The shadow shown in the radiograph of the left side of the chest was thought to be due to consolidation with a small amount of fluid, but subsequent events showed that it was probably almost entirely due to infected fluid. The exploring needle (as almost always) gave more accurate information. No mention is made of any left ventricular hypertrophy (as was indicated by the electrocardiograph), but a moderate amount may well be present without being thought worthy of recording.

6. *Clinical Comments.*—(*a*) General. Whence arose the septic bronchopneumonia? It did not arise from aspiration from the buccal cavity during an operation, for his last operation (that on the tongue) was six weeks previously. Patients succumb much more quickly to septic pneumonia. Whence arose the left-sided empyema? Probably it was a lymphatic spread from the fungating mass on the left side of the neck. How did both lungs become invaded with growth? If through the blood stream, it is remarkable that no other organs were involved. The association of leucoplakia, papillomata and epithelioma in the same tongue is significant. Sampson Handley holds that epithelioma often originates in a papilloma through lymph stasis brought about by obliterative lymphangitis; this may be due to many factors, for example, sepsis, syphilis, mechanical and chemical irritants *et cetera*. There was a history in this case of buccal sepsis, syphilis and heavy smoking.

(*b*) Could a different procedure have saved life? This man came too late for effective treatment. When glandular enlargement is palpable the outlook is not good.

Fracture of the Femur with Diabetes.

1. *Short Clinical History.*—M.L., aged thirty-nine years, a female, was admitted to hospital on March 5, 1934. She died on June 21, 1934. She slipped and fell on the right hip. She was very shocked. She had a fracture in the upper third of the right femur. She had been treated previously for diabetes. The urine, when examined on the day after admission, was found to contain sugar and acetone. Two months after admission she developed pyuria. About a month later she had a temperature of 40° C. (104° F.) with a rigor; prior to this the temperature had been normal; at this time she had a bed sore; from this cellulitis spread to the region around the right ischial tuberosity, accompanied by emphysematous crackling. Her general condition began to get worse. She died two weeks after the onset of cellulitis; the temperature was raised, but the pulse and respiration rates were not appreciably increased.

On her admission to hospital X ray examination showed a pertrochanteric fracture of the femur with impaction; the general alignment was good. Calcification of the femoral artery was noted. Skiagrams taken six weeks later showed union with massive callus.

2. *Account of the Autopsy.*—A large swelling was found over the proximal third of the right femur. Both pleural cavities were obliterated with chronic adhesions. The lungs both showed congestion at the bases. The right was the site of fairly well marked bronchiectasis. In the heart the right atrium was greatly dilated and contained a large amount of *ante mortem* clot, which extended into the pulmonary vein. The heart muscle was very flabby. The coronary arteries and aorta showed marked atheromatous changes. The aorta was very thin at one place. The liver showed chronic venous congestion. Both kidneys were larger than normal and showed marked evidence of acute pyelonephritis. The ureters were thick. The bladder contained pus. The pancreas was smaller and more fibrotic than usual. The brain was normal. The calvarium was much thickened (Figure VI). The right femur manifested a pertrochanteric fracture with a great deal of callus formation around the site of fracture, as is shown in Figure VII. The diameter of this mass, taken obliquely, was ten centimetres. The actual union was not strong; mere handling of the specimen (*post mortem*) was sufficient to cause refracture.

3. *State of the Organs as expected by the Attending Medical Officer.*—Atheroma of the aorta and probably of the coronary arteries was expected, as was some congestion of the lower lobes of both lungs. The urinary tract was thought to be affected by cystitis (secondary to catheterization or to soiling of the parts from incontinence of urine and fæces). The kidneys were not expected to be infected. It was thought that some affection of the islets of Langerhans would be found.

4. *Data on which these Beliefs were Founded.*—Atheroma of the aorta and coronary arteries was suspected because of the marked calcification of the femoral artery as shown in the radiograph. Congestion of the lungs was expected because of long confinement to bed. It was not thought that the infection would ascend from the bladder up to the kidneys, as there was no obstructive lesion. The rigor and high temperature were thought to be due to the cellulitis of the buttock.

5. *Comments as to the Help (or otherwise) of the Tests in this Particular Case.*—The X ray examinations were very helpful.

6. *Clinical Comments.*—(*a*) General. Watson Jones and Roberts,[1] of Liverpool, in a recent article entitled "Calcification, Decalcification and Ossification", claim that incomplete immobilization and immobilization for too short a time are the two main factors responsible for delayed union and non-union; decalcification is caused by the hyperæmia induced by the movements of the part. Conversely, ischæmia causes a recalcification. This progress of events may be seen radiologically during the progress

FIGURE VII.

Excessive callus round a fractured femur in a case of diabetes.

of a fracture; as the early hyperæmic soft tissue passes into the firmer, fibrous, less vascular tissue, so decalcification gives way to recalcification. In another case there was an old infarct in the left ventricle; in this infarct was an area of calcification the size of a Barcelona nut. Ischæmia would be a feature of an organizing blood clot. Thus it is not difficult to understand why thickening of bone and bony outgrowths are a feature, increased calcification or sclerosis being the result of impairment of blood supply—in syphilitic periostitis, in Charcot's disease and, they might have added, in diabetes, in all of which diseases *endarteritis obliterans* occurs.

The thickening of the calvarium in the case of diabetes here described is interesting. I submit the slide showing it (Figure VI).

The frequency of spontaneous fracture in bone which is apparently sclerosed, hard and dense, has not been capable of easy explanation. The significant point is that a dense X ray shadow does not necessarily imply a hard bone.

Marble and chalk cast a shadow of similar density, but the fragility is very different. The resistance of the skeletal framework to injury depends on the resilience of the normal lamellated bone and not on the degree of calcification. When it is recognized that the increased laying down of calcium is evidence of diminished blood supply and reduced vitality, the abnormal fragility is readily understood.

As regards the acute pyelonephritis, was this ascending in nature or was it a blood infection? I am uncertain.

(*b*) Could a different procedure have saved life? She was treated with insulin, diet *et cetera*. I am not aware of any treatment that would have avoided the fatal issue.

Old Pyloric Ulcer Treated by Gastro-Enterostomy; Jejunal Ulcer with Perforation and Repair followed by Stenosis; Recent Gastro-Enterostomy.

1. *Short Clinical History.*—A.A. had had a gastric ulcer for twenty-six years and underwent gastro-enterostomy twelve years ago. Perforation of a gastro-jejunal ulcer occurred eight and a half years ago and was duly repaired. The patient now complained of severe epigastric pain radiating to the back, shoulder and left hypochondrium and aggravated by food; he also had anorexia and occasional vomiting. He was never jaundiced, but lost weight. There were no respiratory or cardiovascular symptoms. Several weeks ago, when he was in the hospital, he became uncontrollable and irresponsible; he rushed into the medical officers' quarters; he was removed to the Reception House.

On examination the patient was found to be very restless and it was difficult to get a history. The temperature, pulse rate and respiration rate were normal. The patient was wasted and the abdomen showed signs of evident gastric peristalsis. The Wassermann test yielded no reaction. On examination the blood, which showed a secondary anæmia, was found to contain 4,310,000 erythrocytes per cubic millimetre, with variation in size and shape; the hæmoglobin value was 46% and the colour index 0·53; leucocytes numbered 8,840 per cubic millimetre; of these, 69% were neutrophile cells, 4% eosinophile cells and 27% lymphocytes.

Administration of a barium meal and subsequent examination revealed no evidence of gastro-enterostomy, apart from some irregularity on the greater curvature. The duodenal cap was very irregular, suggesting either ulcer or adhesions. The lesion was associated with considerable obstruction and peristalsis was marked.

Gastro-enterostomy was performed and the patient died a few hours afterwards.

2. *Account of the Autopsy.*—The most striking thing was the enormous dilatation of the stomach. The original anastomosis was some distance from the pylorus on the greater curvature; it was represented by a mere strand of tissue. The recent one was near the pylorus and further along the jejunum. There was induration externally at the site of the old gastro-enterostomy; internally, a cavity (lined with mucosa), about the size of a plum. The pyloric region was occupied by an indurated mass adherent to the neck of the gall-bladder and surrounding structures. On opening it there was found to be a deep ulcer involving the first part of the duodenum with extensive induration all around it. There was much mucus in the dilated stomach and the duodenum throughout its whole length was considerably dilated. Stenosis at the site of the old gastro-enterostomy was probably the cause of this.

3. *State of the Organs as expected by the Attending Medical Officer.*—In the stomach he expected to find pyloric stenosis with marked dilatation, an old ulcer in the pyloric region, and obliteration of the old gastro-enterostomy.

4. *Data on which these Beliefs were Founded.*—The results of the barium meal examination and the evidence of visible peristalsis were taken as sufficient data.

5. *Comments as to the Help (or otherwise) of the Tests in this Particular Case.*—It would have been helpful if the marked dilatation of the duodenum had also been shown.

6. *Clinical Comments.*—(*a*) General. It is probable that this patient was suffering not only from the symptoms of duodenal ulcer, but from the effects of partial obstruction in two situations: (i) at the pyloric region, (ii) in the jejunum at the site of the old gastro-enterostomy. The effects of acute obstruction involving the upper part of the intestinal tract are known to be very serious; the symptoms of chronic obstruction in the same position are not well recognized. It is quite likely that the symptoms of marked restlessness which were constant in the later weeks and which made the patient sufficiently irresponsible to be taken to the Reception House, were the result of a toxæmia arising from an imperfectly drained duodenum.

(*b*) Could a different procedure have saved life? On the analogy of decompression procedures in the urinary tract, an inlying stomach tube (preferably Levin's, as recommended by Moynihan and used by me on one occasion, which is larger and stiffer than the more commonly used Jutte's, and less readily blocked, size number 7 or 8 English), might be of help in such cases. It is passed through the nose. The stomach can be both fed and washed out with such a tube. It may not be tolerated for more than twenty-four hours at a time, but even that is a great help. It is certainly a matter of common experience that those patients in whom marked dilatation of the stomach is present not infrequently die when a gastro-enterostomy is performed. Possibly prolonged gastric lavage, preliminary to the operation, might serve the purpose. Whatever the best treatment may prove to be, it is certain that any lesion interfering with drainage offends one of the most important principles of surgery. I may be wrong, but I have a feeling that radiologists might more often give us fuller information about the whole duodenum and not merely about the first part.

Pyloric Ulcer with Stenosis; Gastro-Enterostomy.

1. *Short Clinical History.*—J.B., aged sixty-four years, was admitted to hospital on July 3, 1934. He died on July 16, 1934. He suffered from anorexia, vomiting and loss of weight for the last eight months before his death. Electrocardiographic examination of his heart showed left-sided preponderance, and after administration of a barium meal X ray examination revealed pyloric stenosis. Relief of the obstruction was advised by the physician consultant, and on July 14, 1934, a posterior gastro-enterostomy was performed. Two days later the patient died, his temperature being 36·48° C. (97·6° F.), his pulse rate 115, and respiration rate 24.

2. *Account of the Autopsy.*—In the stomach the gastric mucosa and wall were hypertrophied to some degree. There was a simple ulcer present at the pylorus, with much fibrosis, the pyloric canal being practically impassable. Midway along the greater curvature there was a recent gastro-enterostomy, which showed the first signs of union. The pancreas was fibrotic and hard; on section it cut with a grating noise. The kidneys were fairly normal. On examination of the heart, the left ventricle was found to show extreme hypertrophy; the right atrium and right ventricle were considerably dilated, and there was a large amount of *ante mortem* clot in the right side of the heart. The valves, coronary arteries and aorta were normal.

3. *State of the Organs as expected by the Attending Medical Officer.*—In the heart he expected to find some hypertrophy of the left ventricle; in the stomach, pyloric ulcer with accompanying stenosis, some gastric dilatation and hypertrophy.

4. *Data on which these Beliefs were Founded.*—The results of the barium meal examination, the electrocardiograph and the history were considered sufficient data.

5. *Comments as to the Help (or otherwise) of the Tests in this Particular Case.*—The tests were thought to be of very great help.

6. *Clinical Comments.*—(*a*) General. (*b*) Could a different procedure have saved life? It is probable that some of the comments on the previous case apply also to this.

Polycythæmia and Ruptured Duodenal Ulcer.

1. *Short Clinical History.*—J.J., aged fifty-three years, was admitted to hospital on June 22, 1931; he was discharged on July 4, 1931. He was perfectly well one month prior to admission and then began to suffer from severe pain on the right side of the abdomen, not affected by food, and fullness after meals, but no vomiting. He had always had a high colour, but for the last month had been much redder. Nothing particular was noted in the past history. The heart and chest were normal. Distended veins were seen in the lower part of the abdominal wall. The spleen was normal.

The Wassermann test yielded no reaction. A test meal gave a normal result. Venesection was performed, one pint of blood being taken off. The patient was discharged, improved, on July 4, 1931.

A barium meal given on June 23, 1931, revealed a normal stomach with normal evacuation. Slight irregularity of the duodenal cap suggested an ulcer which had not caused any obstruction. The appendix was pathological. The colon was normal.

A blood examination was made on June 6, 1931. The erythrocytes numbered 7,580,000 per cubic millimetre, the hæmoglobin value was 120%, the colour index was 0·8. The leucocytes numbered 14,000 per cubic millimetre. Of these, neutrophile cells were 77·5%, eosinophile cells were 1%, basophile cells 0·5%, and lymphocytes 21%. The red cells were irregular in size and shape.

On June 22, 1934, the erythrocytes numbered 8,400,000 per cubic millimetre, the hæmoglobin value was 171%, the colour index was 1%. The leucocytes numbered 13,000 per cubic millimetre. Of these, neutrophile cells were 84% and lymphocytes 16%. The red cells were irregular.

The patient was readmitted to hospital on July 29, 1934, with a generalized abdominal pain which he had had for some weeks, but which had been much worse during the last twenty-four hours. On examination he was very breathless and cyanosed over the whole of the lower part of the trunk and extremities; the abdomen was extremely rigid and tender. In the chest there were indefinite signs at the bases. His temperature was 38·5° C. (101° F.), his pulse rate was 120 to 140. He died next day; his temperature was then 37·8° C. (100° F.), his pulse rate 100 and his respiratory rate 44. The leucocytes numbered 12,000 per cubic millimetre. The diastatic index was 4. The cause of death was entered as possibly influenzal pneumonia.

2. *Account of the Autopsy.*—The lungs showed extreme congestion. The myocardium was very atonic. The heart valves were normal. The coronary arteries and aorta showed a mild degree of atheroma. All the abdominal organs were extremely cyanosed and the general peritoneal cavity was full of purulent dark fluid. In all quadrants there were extensive adhesions. The spleen was large. The kidneys were congested. There was a perforation in the first part of the duodenum from which gas and intestinal contents were escaping. Longitudinal section of the femur revealed white marrow in the head where normally one expects to get red, and red marrow occupying the length of the shaft where normally one should find white.

3. *State of the Organs as expected by the Attending Medical Officer.*—Duodenal ulcer was expected in the first part, but perforation was not suspected. Some pneumonic consolidation of the lungs and congestion of other organs were expected.

4. *Data on which these Beliefs were Founded.*—The data for these beliefs were a barium meal examination when the patient was in hospital previously, and some doubtful auscultatory signs over the chest behind.

5. *Comments as to the Help (or otherwise) of the Tests in this Particular Case.*—The X ray diagnoses were correct. The bone marrow showed a pathological condition (not yet completely examined or reported upon) which might explain the blood counts.

6. *Clinical Comments.*—(*a*) General. His condition was so bad as to render it difficult to obtain a good history, but the abdominal rigidity was a warning probably of something acute within the abdomen. The fact that he had been in hospital for another condition (polycythæmia) caused the complication of the duodenal ulcer to be overlooked. It is not an infrequent occurrence for one disease to overshadow a second one in the same patient.

A plain skiagram with a portable machine might have revealed gas in the vicinity of the liver. It would not have shown it between the liver and diaphragm, as there were old adhesions.

On auscultation peristaltic sounds over the small intestine area would have been absent. Transmission of heart sounds over the abdomen might have been noted.

With regard to radiography as an aid to the diagnosis of abdominal lesions, I have often advocated a straight X ray examination (that is, one without barium) in cases of possible intestinal obstruction. Normally one sees gas only in the colon; if there is obstruction of the distal part of the colon, then dilatation of the proximal colon is observed.

If a case were one of obstruction in the small intestine, then the colon would be devoid of gas (according to James[2]) and gas and fluid, with straight upper levels, would be seen in the central area of the abdomen (the small bowel area). Lateral as well as antero-posterior pictures are taken in these cases. Of these I have no personal experience.

In addition to radiography we have another aid in auscultation. It is said that in cases of perforation, where there is a mixture of gas and fluid, the heart sounds are transmitted and can be heard through the abdominal wall. Is it because air is a good transmitter?

Auscultation is also valuable in that, if there is paralytic ileus, then the abdomen is silent, free from the gurgling noises normally present. If, on the other hand, there is mechanical obstruction, then the sounds are accentuated. I would make the reservation, however, that I have not found the abdomen quite silent in the early stages of peritonitis. The best place to auscultate is in the lower part of the abdomen.

The presence or absence of liver dulness is often a difficult thing to be sure of. I have always taught that in 50% of cases of perforation there is no perceptible affection of liver dulness (presumably in these cases the perforation is a small one). On the other hand, mistakes may be made in taking liver dulness to be absent because of the distension of the transverse colon or the floating up of a normal one through excessive fluid or blood in the peritoneal cavity. This floating up would be particularly noticeable with a patient in Fowler's position. In a recent case of ruptured liver the apparent absence of liver dulness due to this cause led to the diagnosis of a ruptured hollow viscus.

(*b*) Could a different procedure have saved life? Early recognition and suture of the perforation would probably have saved him.

Abscess of the Liver, with the Formation of Subphrenic Abscess; Pericarditis.

1. *Short Clinical History.*—R.S., aged twenty years, an engineer, was admitted to hospital on July 19, 1934. He died on July 28, 1934. He had had attacks of severe stabbing epigastric pain for seven weeks, becoming progressively worse. Pain was also latterly referred to the left supraspinous region. If the meal was heavy, the pain was worse; otherwise the pain had no relation to meals. The patient had slight pain in the back; the pain was bilateral; there was no vomiting, no jaundice, no cough; and there were no urinary symptoms. The bowel action was regular. He had never been out of Australia and his previous health had always been excellent.

On examination his tongue was furred and dry. His pulse rate was 120; his temperature was 37·8° C. (100° F.). The upper part of his abdomen was rigid. Liver dulness was increased downwards, no tumour was palpable. A laparotomy was performed on July 19, 1934, on the assumption that the condition was a ruptured gastric ulcer. No free fluid or inflammation was present. Stomach, spleen, appendix and gall-bladder were normal. The liver was enlarged, especially the left lobe, which appeared to be

displaced downwards; the post-operative diagnosis was suprahepatic hæmatoma. After operation the pain diminished, but tenderness appeared in the left loin. At this time there were no signs of fluid in the chest. Four days after the operation the general condition was improving. The urine contained a trace of bile, a few red cells and pus cells and some granular casts.

On July 27, 1934, the left pleura was needled and some clear fluid was withdrawn; it set like a jelly before its specific gravity could be taken. The patient died the following day, the temperature having been normal for several days, but the pulse rate high, rising to 130.

The leucocyte count two days prior to operation was 38,000 per cubic millimetre (neutrophile cells 92%); seven days later the count was 23,000; the day following this it was 20,000. The pleural fluid contained 5% protein, thus favouring an inflammatory exudate as against a transudate. It contained a few lymphocytes; there were no tubercle bacilli. The culture was sterile.

2. *Account of the Autopsy.*—In the left lobe of the liver, nearer the posterior surface than the anterior, was a solitary loculated abscess, about the size of a small orange, which had ruptured through into the subphrenic region. The falciform ligament and the inferior *vena cava* formed its right boundary. Adhesions of the liver to the left dome of the diaphragm formed its left boundary. Bacteriological examination of the pus showed a non-hæmolytic streptococcus; no amœbæ were present.

Early fibrinous pericarditis was present. The pericardium was probably infected by lymphatics going through the diaphragm at the inferior *vena cava*. There was nothing particular in the other organs.

3. *State of the Organs as expected by the Attending Medical Officer.*—Enlargement of the liver, especially the left lobe, was expected, but more particularly downward displacement of it, due to a hæmatoma. It was thought that some pleural effusion on the left side would be found.

4. *Data on which these Beliefs were Founded.*—The left lobe of the liver was noticed to be displaced downwards at operation. Fluid was withdrawn from the left pleura.

5. *Comments as to the Help (or otherwise) of the Tests in this Particular Case.*—The progressive fall in the leucocyte count was misleading. The high count and high percentage of neutrophile cells were in favour of an inflammatory focus. The fact that the tests showed the pleural fluid to be inflammatory was some evidence that the infradiaphragmatic lesion causing it was also inflammatory.

6. *Clinical Comments.*—(*a*) General. A single abscess of the liver is uncommon; it sometimes follows (after a variable period) septic infections elsewhere, for example, a carbuncle. There was no history of dysentery (amœbic or otherwise). There was not the usual history of ulcer pain in relation to food; the whole illness was of short duration. Ulcers are not common in one so young. The temperature was normal for several days prior to death; the pulse was a more reliable guide.

In regard to shoulder pain, phrenic referred pain is that pain which is felt on the top of the shoulder as the result of an irritative lesion in the region of the diaphragm. The phrenic is made up of fibres from the third, fourth and fifth cervical nerves, and the referred pain is felt in the region of distribution of the third and fourth, but chiefly the fourth, nerves. Irritation of the right or left side of the diaphragm causes pain which is limited to the shoulder on the corresponding side. Zachary Cope has found that with few exceptions an affection of the anterior part of the diaphragm causes pain in the clavicular region; an affection of the posterior part or crura will lead to local pain in the supraspinous fossa. I do not think that one can discriminate between the upper and under surface of the diaphragm. Cope also found that the phrenic shoulder pain was very common as an accompaniment of a tropical abscess of the liver. It signifies that the liver is adherent to the diaphragm and that the abscess is threatening to break through into the thorax. They usually affect the posterior part of the right lobe.

The abdominal rigidity rightly pointed to the probability of an acute abdominal crisis and justified operation.

(*b*) Could a different procedure have saved life? Possibly early recognition of the liver abscess and the subphrenic abscess with approach from behind might have saved life, but it would have been a fine piece of diagnostic work.

Resection of portion of the twelfth rib (towards its vertebral end) and the use of the sinus forceps would have led to the subphrenic abscess and would have provided excellent drainage without involving the pleural cavity.

Enlargement of the Prostate.

1. *Short Clinical History.*—C.H., aged seventy-one years, complained of weakness in the legs for six months. He had difficulty in micturition for four years. No jaundice, no indigestion, no palpitation, no pain in the chest and no precordial pain were present. Breathlessness was present for six months. The patient complained of syncopal attacks. He denied venereal disease.

On June 5, 1934, he had a catheter tied in the urethra; the residual urine was 480 cubic centimetres (sixteen ounces). On rectal examination the prostate was found to be much enlarged, but did not feel malignant. The urine contained numerous pus cells.

The urea concentration test yielded the following result: Before urea was given, 1·9%; one hour after urea was given, 1·9%; two hours after urea was given, 2·1%; three hours after urea was given, 2·2%. The blood urea nitrogen was 18 milligrammes per 100 cubic centimetres. The blood creatinin was 1·5 milligrammes per 100 cubic centimetres.

The urine water concentration test was made. From 7 p.m. till 7 a.m. the specific gravity of the urine was 1022. At 7 a.m. two pints of water were given. At 8 a.m. the specific gravity was 1012; at 9 a.m. the specific gravity was 1002; at 10 a.m. the specific gravity was 1003; at 11 a.m. the specific gravity was 1005. The whole two pints were passed. The rising concentration and the passage of the whole amount of the water indicated good function.

The indigo-carmine test was performed. Eight cubic centimetres of 0·4% indigo-carmine were injected intravenously. The dye first appeared in six minutes. The urine was pale blue in nine minutes. It was dark blue in eleven minutes.

X ray examination showed the kidneys to be normal. Radiological examination also revealed some well marked enlargement of the aorta with some calcification of its walls. Left ventricular hypertrophy was present. The electrocardiograph revealed some left ventricular preponderance, but no other abnormality. Thus the renal functions were good, but there was some doubt about the circulatory system. The pulse was weak.

On June 19, 1934, prostatectomy was performed under spinal anæsthesia. The operation took an hour, was very difficult, especially the separation of the left lobe. Much hæmorrhage occurred. The patient was returned to the ward very shocked; he died next day. He was thought to have a left cerebral hæmorrhage. The right pupil was pinpoint, the left pupil was dilated. The right arm and leg were flaccid. The right knee jerk was exaggerated; the toe reflex was extensor. The left arm and leg were spastic. The temperature was normal all the time the patient was in hospital, until the day of death, when it was 38·9° C. (102° F.).

2. *Account of the Autopsy.*—The left ventricle was hypertrophied; the aorta and mitral valves were thickened. The coronary arteries were very atheromatous. Extensive atheroma of the aorta was present, both the white syphilitic type and the yellow ordinary type. Except for some pallor and excess of fat in the pelvis, the kidneys were normal. The abdominal glands were not enlarged. The bladder contained a moderate amount of blood. The brain showed no sign of hæmorrhage or other lesion.

3. *State of the Organs as expected by the Attending Medical Officer.*—In the heart hypertrophy of the left ventricle was expected, with some myocardial degeneration due to coronary atheroma. It was thought that atheroma of the aorta would be found, that some interstitial changes would be present in the kidneys, and that a hæmorrhage would be present in the left cerebral hemisphere involving the pyramidal tracts.

4. *Data on which these Beliefs were Founded.*—The data for these beliefs were: the history of breathlessness, the X ray findings and the electrocardiographic findings. The result of the urea concentration test indicated some slight renal degeneration. The right-sided flaccidity and exaggeration of the knee jerk, together with the extensor response of the great toe, seemed to indicate an affection of the left pyramidal tract, probably a hæmorrhage from its rapid onset.

5. *Comments as to the Help (or otherwise) of the Tests in this Particular Case.*—The X ray findings, the electrocardiographic findings and the results of the renal tests were very helpful. The interpretation of the neuromuscular signs was somewhat astray. The prostate was wrongly assumed to be adenomatous, but was rightly interpreted as non-malignant.

6. *Clinical Comments.*—(*a*) General. I think death resulted from shock, accentuated by hæmorrhage.

(*b*) Could a different procedure have saved life? Possibly cutting portion of the prostate by the diathermy knife (intraurethral electrode) might have been better, if it had been realized that enucleation was going to be so difficult.

Fracture of Tibia and Fibula.

1. *Short Clinical History.*—R.H., aged eighty-three years, was admitted to hospital on July 8, 1934. He died on July 22, 1934. He fell down stairs, sustaining a fracture of the right leg. X ray examination several days later revealed a fracture of the middle third of the right tibia and of the right fibula near the neck and at the malleolus. As the position was satisfactory, the leg was put up in plaster, without an anæsthetic. Ten days after admission to hospital he was not so well. His temperature, pulse and respiration, which had been respectively 37·1° C. (98·8° F.), 100 and 22 on admission, gradually rose seven to ten days after admission until the fourteenth day, when they were 39·4° C. (103° F.), 160 and 48. He then died. Death was certified as due to fracture of the tibia and fibula, shock and cardiac failure.

2. *Account of the Autopsy.*—He was a wasted old man. In the chest pleural adhesions were present on both sides. The lungs showed very marked anthracosis and intense congestion at the bases. Slight dilatation of the right auricle and ventricle was present. The mitral valves were slightly thickened; marked calcification of the aortic cusps was present; the cusps were quite competent. A very occasional patch of atheroma was found in the aorta and coronary arteries. The liver was shrunken and showed evidence of chronic venous congestion. There were about fifty gall-stones present in the gall-bladder. The spleen was small. The kidneys both contained many cysts, varying from a pin's head to seven centimetres in diameter. Definite evidence of interstitial nephritis was present. The sigmoid colon was adherent to the bladder and felt very hard; in places diverticulosis was present, and in others diverticulitis with much fibrosis of the wall and narrowing of the lumen. In the right leg there was a fracture of the tibia and fibula with much effusion of blood into the deep muscles. The right knee joint showed evidence of osteoarthritis, the cartilage of the femur and tibia being eroded in places. There was a loose body in the knee joint.

3. *State of the Organs as expected by the Attending Medical Officer.*—Hypostatic pneumonia of the lungs was suspected, also some probable interstitial nephritis. Atheroma of the aorta and coronary vessels was also suspected.

4. *Data on which these Beliefs were Founded.*—These beliefs were largely clinical surmise based on the extreme age of the patient. The raised temperature, pulse and respiration rate were thought to be due to hypostatic pneumonia.

5. *Comments as to the Help (or otherwise) of the Tests in this Particular Case.*—The X ray findings were accurate as regards the leg. No other tests were performed.

6. *Clinical Comments.*—(*a*) General. The autopsy seems to show that uræmia was the probable cause of death, and not heart failure. Presumably the accident, with its attendant shock, was sufficient to tip the scale and to interfere with the function of the kidneys, which had very little reserve.

Watson Jones and Roberts[1] point out that patients whose treatment demands prolonged recumbency and immobility frequently develop large numbers of calculi in the renal pelvis and ureter; generalized disease depletes the calcium store of the whole skeleton, in addition to the local decalcification of the affected bones (as in fracture). They go on to point out that, quite apart from the risk of renal inefficiency due to obstruction, the prolonged excessive excretion of calcium in itself may lead to renal cellular degeneration and retention of non-protein nitrogen. That this was a factor sufficient to turn the scale in the present case I do not believe, for, even allowing for the marked degeneration of the kidneys, the period of recumbency and disease was very short. Neither heart nor lungs seemed primarily responsible for death. The presence of so many pathological lesions was the penalty of living to eighty-three. There was no common ætiological factor in the nephritis, the diverticulitis, the gall-stones, the osteoarthritis.

(*b*) Could a different procedure have saved life? Nothing could have saved the patient.

Conclusion.

Of these ten patients only one (the patient with the epithelioma of the tongue) had been under my care, prior to my resigning from the active staff of the Sydney Hospital, so that the clinical remarks are such as I assume might have been made by the surgeon in charge of the case.

Acknowledgements.

For the microphotographs and lantern slides I am indebted to the Pathological Department of Sydney Hospital, the Director, Dr. Keith Inglis, and Miss Jones, the clinical photographer attached to the department.

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THE TASMANIAN CRANIA IN COLLECTIONS IN THE COMMONWEALTH.[1]

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I have thought it advisable to treat the subject in a broad, general way, because of limited time. Detailed particulars will be provided in an article of which this paper is practically a summary. The article will be a record of work done in recent years with the aim of obtaining a reliable craniological description of the skull of the Tasmanian full-blood.

Skulls of any kind are interesting to some people, but there are several reasons why the Tasmanian skulls are of special interest. They are as follows:

[1] Read at a meeting of the Australian and New Zealand Association for the Advancement of Science, Melbourne, January, 1935.

1. The Tasmanian's origin is still a mystery, and the skulls may hold the secret to its solution. Several theories have been advanced about this question, but none has been satisfactorily proved or disproved so far.

2. The data to be obtained from them may help in clearing up the question of whether the Tasmanian inhabited the mainland and formed a link in the ancestral line of the Australian.

3. Their scarcity. The Tasmanian race became extinct about sixty years ago, and the cranial remains in the Commonwealth number only 87 specimens in public collections and 18 in private collections, making a total of 105 which are claimed to be Tasmanian. In overseas collections there are only about 80 specimens.

4. Very little reliable information was left by those who observed the living Tasmanians, and consequently the skulls and the cultural remains constitute the only sources from which any can be gathered now.

5. As this is a science congress, we are not concerned with the monetary value of these skulls, which is the only interest for some people.

It was found to be necessary to inquire into the various examinations of these skulls that have been made up to the present time, in order to obtain a basis for testing the theories about the Tasmanian's origin. This inquiry revealed some surprises.

The first systematic investigation was made in 1897 by Harper and Clarke,[1] who examined 18 specimens in the Hobart museum. They provided measurements and also information regarding characteristics which cannot be measured. The methods used by them were sound, their descriptions clear and definite, and the results are regarded as reliable (one of these authors was Dr. A. H. Clarke, Hobart, whose father interpreted the treaty of Waitangi in 1840 between the British Government and the Maoris).

The second examination was made in 1909 by Berry and Robertson[2] and several associates. The two principals claimed credit for discovering many skulls, which, added to the 18 examined by Harper and Clarke, brought the total to 52. This "discovery" was due largely to much unacknowledged help from others, notably Dr. W. L. Crowther, of Hobart. Several inconsistencies and faults have been found in connexion with the methods they employed, and many errors, some as great as 10%, have been discovered in their records. The mathematical work of K. S. Cross,[3] of Melbourne is beyond reproach, but the data supplied to him by the leaders of the team were undoubtedly faulty. The whole of their work refers only to the form and size of the skull. Its value is therefore limited. The results of their investigation, judged by the inquiry referred to, do not provide a reliable description of the Tasmanian skull.

The third examination was made by Ramsay Smith in 1910. He measured two skulls and a fragment in the Sydney museum, and one skull in a private collection which has since been lost. In addition to the measurements he gave a description of many non-metrical characteristics.

The fourth investigation was made in 1924 by Wood Jones and Campbell, who described six specimens in the Adelaide museum. This was the first occasion on which Tasmanian skulls had been measured according to the directions advocated by the International Agreement of 1906. The inquiry revealed that this work and that of Harper and Clarke are far superior to the works of the other investigators up to 1924.

Various examinations of single specimens were made by different workers, but were not carried out systematically.

Under the general direction of Professor Wood Jones, the fifth investigation was made by me in 1931. All the skulls in the Commonwealth claimed to be Tasmanian were examined in the following way: (i) Forty-one measurements, as outlined by the International Agreement, were made on each skull, or as many of them as the condition of the skull allowed. (ii) A search was made on each skull for twenty-six characters which had been chosen by Wood Jones[4] for use for racial diagnosis.

When the results of all these examinations were analysed, the product was chaotic. One fact which stood out in clear relief was that if the majority of the skulls were Tasmanian, the remaining minority were not. Consequently I started last year to make a general survey of the whole series and to check my own and all the other workers' measurements and descriptions. Unfortunately this survey is not yet completed.

Reference to any historical work on Tasmania reveals that the world was not much better in some respects in 1835 than it is today. The half-caste problem had already begun. It is therefore extremely unlikely that all the skulls found on the island are actually those of full-blood natives.

The Authenticity of the Specimens.

The problem of ascertaining which specimens are authentic then arose. How was this to be done at such a late date, seeing that the history of only a few of the skulls was known? Some criterion had to be chosen.

Reference was made to the work of Sir William Turner, who supplied a clear and definite description of the Tasmanian's skull. His description contains some thirty-six general characteristics which he regarded as specifically Tasmanian, and it was decided to use them as a standard for comparative purposes. Turner gave sound reasons for regarding some skulls in English collections as not those of Tasmanian full-bloods, although labelled "Tasmanian". He classified some as those of mixed-bloods, others as Polynesian, and at least one as European.

Going back to the examinations made by Harper and Clarke, Berry and Robertson, and Ramsay

Smith, it was found that all referred to Turner's work and agreed with him that Tasmanian skulls exhibit a remarkable approach to uniformity of form and size. However, the only investigators who showed an intelligent grasp of Turner's work, and also ability to apply his findings with discrimination, were Harper and Clarke, who grouped the 18 specimens in the Hobart museum as 12 Tasmanian full-bloods, 3 Tasmanian mixed-bloods, 3 without any trace of Tasmanian origin.

In marked contrast is the attitude of Berry and Robertson, who regarded every skull submitted to them as that of a Tasmanian full-blood. They did not raise a doubt about even one specimen in the 52 they examined. They placed implicit faith in the labels attached to the skulls—a very unwise reliance, according to Turner. Worse still, they offered very unconvincing reasons for doubting the judgement of Harper and Clarke, and Berry even went so far as to interview Clarke to try to convince him that he had made an error. The attitude of Berry and Robertson was not one of scientific inquiry.

The use of Turner's description in the present survey has resulted in the classification of Harper and Clarke being generally confirmed, while only about 33 of the 52 specimens examined by Berry and Robertson are regarded as those of Tasmanian full-bloods.

In this part of the survey an attempt has been made to answer three questions in an endeavour to determine whether a skull is authentic or not. They are as follows: (i) Is the skull that of an aboriginal? (This is to eliminate European skulls.) (ii) Is the skull that of a mixed-blood? (iii) Is the skull that of a Tasmanian full-blood?

Roughly 80% of the total of 105 specimens have been classified as pure Tasmanian, the remainder consisting chiefly of Tasmanian-white mixed-bloods, a few pure Australians, and mixed-bloods of races other than the Tasmanian.

In the present survey each skull must pass a test containing over one hundred particulars.

Historical records and narratives provide abundant evidence of the admixture of black and white blood after the beginning of white settlement and, to a less extent, in the earlier days of the sealers and whalers. There were also official and unofficial transportations of natives to and from the mainland. My interest in the historical aspect of this matter was aroused by Mr. T. Dunbabin, M.A., of Sydney.

When examining Maori skulls in New Zealand, Professor J. H. Scott found that many had to be excluded because they showed traces of white admixture or were not of Maori origin.

The differences between the authentic and unauthentic specimens are more clearly revealed by illustrations than by verbal descriptions. Only a few of the specific Tasmanian characters can be shown in the time available.

Figure I shows a skull viewed from above and regarded by Turner as that of a male Tasmanian full-blood. It shows the pentagonal outline, the great width between the parietal eminences, and the

FIGURE I.

relatively narrow frontal width. In masculine skulls there is usually a distinct triangle on the frontal bone, with the base on the coronal suture.

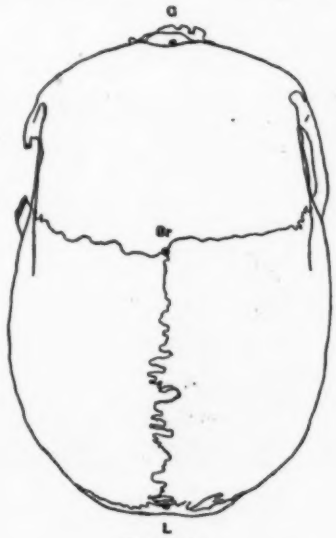

FIGURE IA.

Note the contrast in outline shown in Figure IA, which is seen in a few of the specimens. The difference between the two outlines may be regarded as a measure of the difference between all the other

characteristics of form exhibited by these skulls and those of the full-bloods. This outline is typically Australian. Many Australian natives were transported to Tasmania and some died there.

Figure II is a side view of the skull shown in Figure I. The outline is regularly curved, and the surface of the bone is seen to be very smooth, even the areas of muscle attachment being only a little

Figure II.

rougher. The brow region is very prominent and the root of the nose is deeply depressed. All ridges and borders are rounded. The orbit is peculiar. The upper border is in front of the lower and the outer margin is far behind the inner.

The skull shown in Figure IIa exhibits many contrasts. It is so heavy and the surface is so rough that, when handled, it gives the impression of

Figure IIa.

having been carved out of stone. All its ridges and borders are sharp. It has a tell-tale elevation on the frontal bone in front of the bregma. Evidence of white admixture in the cranial part of these skulls is usually noticeable on the frontal bone. The orbit of this skull is very different from that of the full-blood specimen, the relative positions of the borders being reversed. The inion is large. The malar bone is coarse and thick and is situated further anteriorly than in the Tasmanian. The jaw is misshapen, the dental arch is irregular and narrow, and the teeth are of the degenerate kind seen in Europeans. Harper and Clarke regarded this skull as that of a Tasmanian half-caste. Berry and Robertson were satisfied that it was that of a Tasmanian full-blood. In the present survey it is classified as definitely that of a mixed-blood, but not Tasmanian. It is believed to be that of a Polynesian mixed-blood, probably a Maori half-caste.

The skull shown in Figure III is the same as that in Figures I and II.

The vault is "roof-shaped" and the centre line is "keeled". Both these terms were used by Turner in referring to the vault. I have suggested a modi-

Figure III.

fication of Turner's description of the keeling, which, I find, is limited to the anterior one-third of the sagittal suture, while the middle and posterior one-third are depressed between two lateral ridges. This helps to distinguish the Tasmanian from the Australian skull, the keeling in the latter extending further posteriorly. The orbits are rectangular; this is seen better in the actual skull, because they are set obliquely. The width of the nasal aperture is great, exceeding in many specimens half the height. The nasal spine is not prominent. The jaws and dental arches are wide and regular in shape. The teeth are not of the degenerate kind.

The differences seen in the skull shown in Figure IIIa are very distinct. The orbits are more nearly square. The nasal aperture is very narrow and high. The nasal spine is prominent. The jaw

is misshaped, the dental arch very narrow and irregular, and the teeth are like those seen in the mouths of Europeans.

The chief differences between the skulls of Tasmanian full-bloods and those of mixed bloods are seen in the facial part, only minor differences being exhibited in the cranial part. This fact seems to have caused many investigators to regard all skulls with a general resemblance in the cranial part to that of the pure Tasmanian as authentic, the evidence to the contrary exhibited in the facial part being entirely overlooked.

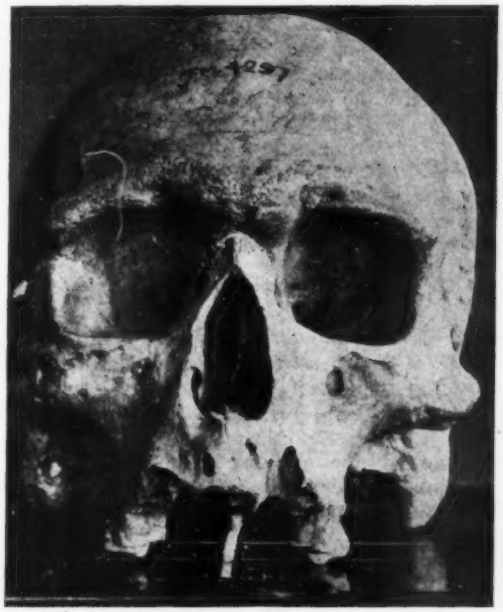

Figure IIIa.

The skull shown in Figure IV is that of the well known Truganini, the last of her race. The keeled vault, the narrowness of the forehead, compared with the maximum cranial width and the wide nasal aperture are apparent.[1]

The specimen illustrated in Figure IVa has been described by one investigator as that of a female Tasmanian full-blood. It will be noted that the width of the forehead is little less than the maximum cranial width. The width of the nasal aperture, compared with the height, is much less than usual, although the lateral margins have been broken. In the present survey this specimen has been classified

Figure IV.

as definitely that of a mixed-blood, and more probably an Australian-white mixed-blood.

Figure IVa.

Figure V is a specimen greatly damaged by fire. Berry and Robertson recorded its measurements, but excluded them in calculating indices because it

[1] Note that this skull is charred. Many of the specimens are in a similar condition owing to the burial rites of the natives, but it puzzled me to find that the skull of the last survivor is not an exception. Mr. A. L. Meston, M.A., of Launceston, has informed me that residents in the locality where Truganini died were aware that, before death, she dreaded the possibility that her body would ultimately be cut up for scientific purposes. As a mark of respect for her, and to prevent this possibility, they did not incinerate her body, but boiled it in a large copper, the resulting stench permeating the atmosphere of the neighbourhood throughout a whole week. Her skull, seen in the Hobart museum today, reveals that all trace of the charring has been removed in the meantime by sandpapering, with consequent reduction of its value scientifically.

is that of a juvenile. In the present survey it has not been measured because it is in a soft condition, anatomical points are obscured, and racial diagnosis is impossible. Because a desiccated brain was found in this skull, it is probably valuable ethnologically, but it is valueless for craniometrical purposes.

Figure V.

Two cranial fragments are shown in Figure VI. Their measurements were recorded by Berry and Robertson, who regarded their size and condition as suitable for providing reliable craniometric data. In the present survey measurements of these specimens have been excluded, principally because they are too fragmentary to enable a reliable diagnosis to be made or to provide any data of value.

Figure VI.

Some Unique Specimens.

Some skulls and limb bones were found at Eaglehawk Neck on the east coast of Tasmania a few years ago. They are the remains of some male and female adults and children who evidently perished together. Various explanations have been advanced to account for their death, such as an epidemic disease, a tidal wave or lightning. There is no evidence to show that they were killed by fighting.

The skulls and other bones indicate that they belonged to natives much taller than the ordinary Tasmanians. No trace of racial admixture has been found. The examination of them is not complete, but it points to the probability that they are the remains of a tribe of very tall natives or else they represent some of the ancestral members of the Tasmanian race.

Part of a skull found not far distant on the same coast is extremely thick. The thickness varies from a little over 1·25 centimetres (half an inch) to a little under 1·8 centimetres (three-quarters of an inch). It has several specific Tasmanian characters and formed part of a large skull closely resembling those just referred to. It does not exhibit any evidence of mineral deposition or any pathological condition. The maximum thickness of Tasmanian skulls is usually not greater than 1·25 centimetres (half an inch).

Bones are not usually amusing, but one skull proved an exception. It is that of a Tasmanian full-blood who, in life, was presumably black. This specimen was coated with paint of flesh pink colour. It possessed about six teeth, which belonged to it, while the rest were obviously Australian teeth, inserted into the sockets by someone with more enthusiasm than knowledge. Some of them had been glued to the wrong side of the jaw, while others were back to front. The effect can be imagined.

Regarded seriously, such treatment of a scientific specimen reduces a museum to the level of a wax-works and should be condemned.

Summary.

1. The Tasmanian skulls are of interest because they are closely connected with the questions of how the natives reached Tasmania and whether the Tasmanians inhabited the mainland.
2. The skulls are scarce and should receive proper protection as scientific specimens.
3. Some of the various examinations can be relied upon, while others cannot. The one which should be given premier position is that of Harper and Clarke.
4. The skulls of the Tasmanian full-bloods are remarkably alike. A very lucid description of them was given by Turner. Any trained observer, knowing Turner's work, should not have difficulty in eliminating unauthentic specimens. About 20% of the skulls labelled as Tasmanian are regarded as unauthentic.
5. Conclusions will not be justified until the present work is completed. However, one is justified in saying that there is no anatomical evidence to support the statement so often seen in books on Tasmania, that the natives were very wild, low-grade savages with enormous jaws and teeth. The Tasmanian was not vigorous, for a primitive man, and his jaws and teeth are smaller than the Australian's and very different in many other respects.

Conclusion.

Before concluding I should like to make a few remarks.

Any criticism I have given has been made in the interests of craniology and has no personal aspect.

The statement has been made at a meeting of this congress that those who work on skulls consider that their field is more important than any other in anthropology. This criticism, however, does not apply to some of us.

ILLUSTRATIONS TO THE ARTICLE BY DR. H. S. STACY.

FIGURE III.

Skiagram showing calcified arteries of the arm in a case of diabetes.

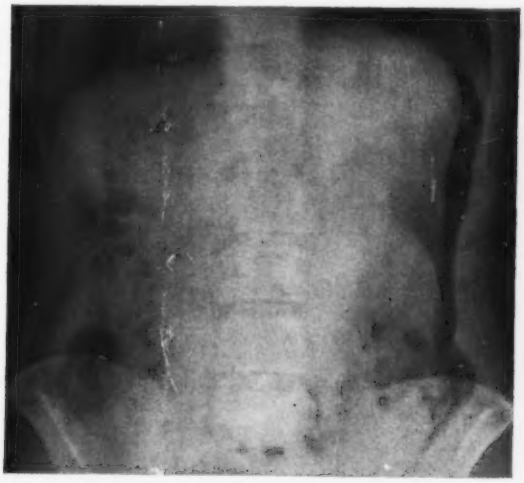

FIGURE IV.

Skiagram of pneumoperitoneum. In this instance only 360 cubic centimetres of air were injected; this amount was insufficient. The layer of air can be seen over the right lateral surface of the liver.

FIGURE V.

Skiagram of pneumoperitoneum differentiating shadow of carcinoma of the lung from adjoining liver shadow. The air can be clearly seen below the right dome of the diaphragm. (By courtesy of Dr. Cotter Harvey and Dr. Vickery.)

FIGURE VI.

Thickened calvarium in a case of diabetes.

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My own view is that we should try to make a contribution towards the fund of knowledge about the aboriginal that will command the respect of fellow workers in other fields. Such a contribution should pass the most searching inquiry and is of value only if it is related to contributions from other fields. Beyond that, we should encourage rather than try to belittle the work of others.

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THE USE OF A PERMANENT STANDARD IN THE FOLIN-WU METHOD OF DETERMINATION OF SUGAR IN BLOOD.

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THE method of Folin and Wu for determining the blood sugar content has the great merit of simplicity *plus* a degree of accuracy that compares favourably with any other standard method of estimation. One of the objections of the method, to my mind, is the necessity for using a weak glucose solution as a standard. The standard, in spite of the use of preservatives, is liable to undergo rapid deterioration, and unless the worker has sufficient knowledge of the method, he may not realize that the glucose content of the standard has diminished considerably.

Having this in view, I have worked for a considerable time in an endeavour to establish a permanent standard for colorimeter work. After hundreds of estimations and comparisons of some results with Hagedorn and Jensen's micro-method of determination of blood sugar, I evolved a technique for three fixed standards for insertion in the colorimeter. These were prepared in conjunction with Hellige and Company, Freiburg; they are as follows:

Standard I, for readings between 60 and 125 milligrammes of glucose per 100 cubic centimetres of blood.

Standard II, for readings between 125 and 250 milligrammes of glucose per 100 cubic centimetres of blood.

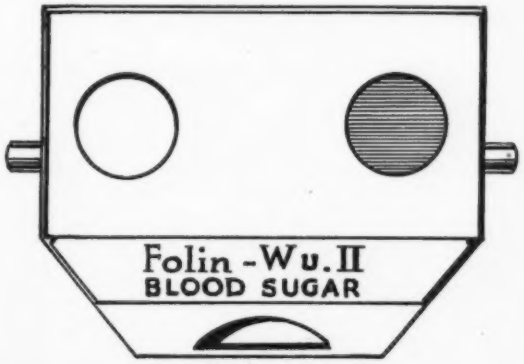
Standard III for readings between 225 and 500 milligrammes of glucose per 100 cubic centimetres of blood.

These standards were prepared from a non-fading glass, inserted into a vulcanite frame for placing into the aperture under the eyepiece of the colorimeter.

Direction for Determination of Sugar in Blood (Folin-Wu Method) with Hellige Colorimeter and Glass Standard.

First adjust the colorimeter reflectors so as to produce uniform illumination in both half fields; be sure also that the colorimeter has been tested so that zero readings on both sides agree.

Insert the blood sugar standard underneath the prism housing (remove the right-hand cup and plunger). Care should be taken to see that the standard is properly inserted, not reversed.



Method.—Place in a centrifuge tube: (*a*) 0·7 cubic centimetre of distilled water; (*b*) 0·1 cubic centimetre of blood, obtained by pricking the lobe of the

ear, finger *et cetera*; (*c*) 0·1 cubic centimetre of 10% sodium tungstate solution; (*d*) 0·1 cubic centimetre of 2/3 N. sulphuric acid.

Mix thoroughly with fine glass rod.[1] If the Folin improved protein precipitant reagent is used, the following quantities are taken: (*a*) 0·8 cubic centimetre of improved reagent, (*b*) 0·1 cubic centimetre of blood, (*c*) 0·1 cubic centimetre of 1/3 N. sulphuric acid.[2] Centrifuge for three minutes. Transfer 0·5 cubic centimetre of the clear supernatant liquid to a Folin sugar tube, graduated at 6·25 cubic centimetre and constricted at the 1 cubic centimetre mark instead of the usual 4 cubic centimetre of the 25 cubic centimetre sugar tube. Add 0·5 cubic centimetre of the alkaline copper tartrate reagent. The tube is then placed in the boiling water bath for six minutes. Add 0·5 cubic centimetre of phosphomolybdic acid reagent and dilute the resulting blue solution to the 6·25 cubic centimetre mark with distilled water. Close the sugar tube with the thumb, mix well by inverting slowly four or five times so that the blue colour is distributed uniformly.

Simple inspection of the unknown blood sugar will indicate which standard to use. Place the coloured solution in a colorimeter cup and insert the latter on the left-hand platform of the colorimeter. After determining the colorimeter reading the blood sugar values are then read from the appropriate tables.

Table I.

For Standard Sugar in Blood.

Liquid Depth. Millimetres.	Milligrammes of Sugar in 100 Cubic Centimetres of Blood.	Liquid Depth. Millimetres.	Milligrammes of Sugar in 100 Cubic Centimetres of Blood.	Liquid Depth. Millimetres.	Milligrammes of Sugar in 100 Cubic Centimetres of Blood.
10·0	125·3	13·7	91·4	17·4	71·9
10·1	124·0	13·8	90·8	17·5	71·5
10·2	122·8	13·9	90·1	17·6	71·1
10·3	121·6	14·0	89·4	17·7	70·7
10·4	120·4	14·1	88·8	17·8	70·3
10·5	119·3	14·2	88·2	17·9	69·9
10·6	118·2	14·3	87·6	18·0	69·5
10·7	117·1	14·4	87·0	18·1	69·1
10·8	116·0	14·5	86·4	18·2	68·8
10·9	114·9	14·6	85·8	18·3	68·4
11·0	113·9	14·7	85·2	18·4	68·1
11·1	112·8	14·8	84·6	18·5	67·7
11·2	111·8	14·9	84·1	18·6	67·3
11·3	110·8	15·0	83·5	18·7	66·9
11·4	109·9	15·1	82·9	18·8	66·6
11·5	108·9	15·2	82·4	18·9	66·2
11·6	108·0	15·3	81·9	19·0	65·8
11·7	107·1	15·4	81·3	19·1	65·5
11·8	106·1	15·5	80·8	19·2	65·2
11·9	105·3	15·6	80·3	19·3	64·8
12·0	104·4	15·7	79·8	19·4	64·5
12·1	103·5	15·8	79·3	19·5	64·2
12·2	102·7	15·9	78·8	19·6	63·9
12·3	101·8	16·0	78·3	19·7	63·5
12·4	101·0	16·1	77·8	19·8	63·2
12·5	100·2	16·2	77·3	19·9	62·9
12·6	99·4	16·3	76·8	20·0	62·6
12·7	98·6	16·4	76·3	20·1	62·3
12·8	97·9	16·5	75·9	20·2	62·0
12·9	97·1	16·6	75·4	20·3	61·7
13·0	96·3	16·7	75·0	20·4	61·4
13·1	95·6	16·8	74·5	20·5	61·1
13·2	94·9	16·9	74·1	20·6	60·8
13·3	94·2	17·0	73·7	20·7	60·5
13·4	93·5	17·1	73·2	20·8	60·2
13·5	92·8	17·2	72·8	20·9	59·9
13·6	92·1	17·3	72·3	21·0	59·6

Table II.

For Standard Sugar in Blood.

Liquid Depth. Millimetres.	Milligrammes of Sugar in 100 Cubic Centimetres of Blood.	Liquid Depth. Millimetres.	Milligrammes of Sugar in 100 Cubic Centimetres of Blood.	Liquid Depth. Millimetres.	Milligrammes of Sugar in 100 Cubic Centimetres of Blood.
7·5	302	11·4	199	15·3	148
7·6	298	11·5	197	15·4	147
7·7	294	11·6	195	15·5	146
7·8	290	11·7	193	15·6	145
7·9	287	11·8	192	15·7	144
8·0	283	11·9	190	15·8	143
8·1	280	12·0	189	15·9	142
8·2	276	12·1	187	16·0	141
8·3	273	12·2	186	16·1	140
8·4	269	12·3	184	16·2	139
8·5	266	12·4	183	16·3	139
8·6	263	12·5	181	16·4	138
8·7	260	12·6	180	16·5	137
8·8	257	12·7	178	16·6	136
8·9	255	12·8	177	16·7	135
9·0	252	12·9	176	16·8	134
9·1	249	13·0	174	16·9	133
9·2	246	13·1	173	17·0	133
9·3	244	13·2	172	17·1	132
9·4	241	13·3	170	17·2	131
9·5	238	13·4	169	17·3	130
9·6	236	13·5	168	17·4	130
9·7	233	13·6	167	17·5	129
9·8	231	13·7	165	17·6	128
9·9	229	13·8	164	17·7	127
10·0	227	13·9	163	17·8	127
10·1	224	14·0	162	17·9	126
10·2	222	14·1	161	18·0	125
10·3	220	14·2	160	18·1	125
10·4	218	14·3	158	18·2	124
10·5	216	14·4	157	18·3	123
10·6	214	14·5	156	18·4	123
10·7	212	14·6	155	18·5	122
10·8	210	14·7	154	18·6	121
10·9	208	14·8	153	18·7	121
11·0	206	14·9	152	18·8	120
11·1	204	15·0	151	18·9	120
11·2	202	15·1	150	19·0	119
11·3	200	15·2	149		

Table III.

For Standard Sugar in Blood.

Liquid Depth. Millimetres.	Milligrammes of Sugar in 100 Cubic Centimetres of Blood.	Liquid Depth. Millimetres.	Milligrammes of Sugar in 100 Cubic Centimetres of Blood.	Liquid Depth. Millimetres.	Milligrammes of Sugar in 100 Cubic Centimetres of Blood.
12·0	510	15·4	366	18·8	296
12·1	503	15·5	363	18·9	294
12·2	497	15·6	360	19·0	292
12·3	491	15·7	357	19·1	291
12·4	486	15·8	355	19·2	289
12·5	480	15·9	352	19·3	288
12·6	474	16·0	350	19·4	286
12·7	469	16·1	347	19·5	285
12·8	464	16·2	345	19·6	284
12·9	459	16·3	343	19·7	282
13·0	455	16·4	340	19·8	280
13·1	450	16·5	338	19·9	279
13·2	446	16·6	335	20·0	278
13·3	441	16·7	333	20·1	277
13·4	436	16·8	331	20·2	275
13·5	432	16·9	329	20·3	274
13·6	428	17·0	327	20·4	272
13·7	423	17·1	325	20·5	271
13·8	419	17·2	323	20·6	270
13·9	415	17·3	321	20·7	268
14·0	411	17·4	319	20·8	267
14·1	407	17·5	317	20·9	265
14·2	404	17·6	315	21·0	264
14·3	400	17·7	314	21·1	263
14·4	397	17·8	312	21·2	262
14·5	394	17·9	310	21·3	260
14·6	390	18·0	308	21·4	259
14·7	387	18·1	307	21·5	258
14·8	384	18·2	305	21·6	256
14·9	381	18·3	303	21·7	255
15·0	378	18·4	302	21·8	254
15·1	375	18·5	300	21·9	253
15·2	372	18·6	299	22·0	251
15·3	369	18·7	297		

Acknowledgement.

I wish to thank Dr. E. B. Jones, of the Prince Henry Hospital, for assistance that he has given me in some of the experimental work.

Summary.

A new method of using a permanent standard for reading blood sugar estimations up to 500 milligrammes per 100 cubic centimetres by colorimeter is described, which for all times insures a permanency of standards and a quick and accurate method of determining blood sugars.

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Reports of Cases.

DEFICIENT FIXATION WITH PROBABLE VOLVULUS OF THE MID-GUT LOOP IN AN INFANT.

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Clinical History.

Baby N.D., a male, aged six days, was admitted to the Royal Alexandra Hospital for Children, Sydney, on November 16, 1934. The confinement had been normal and the infant appeared to be a normal healthy boy. The birth weight was 3·6 kilograms (eight pounds). Meconium was passed as usual, and for the first four days the stools were normal. On the fifth day he commenced to vomit the breast milk, and the vomitus soon became bile-stained and the vomiting was projectile. The stomach was washed out, but the vomiting became more frequent and the vomitus more bile-stained. There was no generalized abdominal distension, but the distended stomach could be readily seen in the epigastric region. There was no visible gastric peristalsis. The lower part of the abdomen was flattened. The child began to lose weight rapidly. It was obvious that there was some duodenal obstruction.

The stomach was washed out and a large quantity of bile-stained fluid was evacuated. The abdomen was opened through a mid-line incision, about 6·75 centimetres (two and a half inches) long, with the umbilicus at its centre. The disposition of the viscera appeared to be normal. There was no evidence of non-rotation or reversed rotation of the mid-gut loop. The whole of the small bowel was collapsed and empty. The large bowel was smaller than normal and apparently empty. The transverse colon was withdrawn and turned upwards on to the abdominal wall, and the whole of the small bowel was withdrawn through the wound. The mesentery had only a narrow pedicle instead of the usual broad attachment, and its base was thickened and œdematous. No actual volvulus could be detected. The duodenum was fixed in its normal position, but appeared to be slightly dilated. There was a band of tissue stretching between the last five centimetres of the duodenum and the first five centimetres of the jejunum. This band caused a kinking of the duodeno-jejunal flexure. A fossa was thus formed, with its opening pointing downwards and to the right, and into which a finger could be passed. The tissue forming the anterior wall of this fossa was divided and the bowel was returned into the abdomen. The child had no further vomiting and made an uneventful recovery.

Commentary.

This case was one in which there was some derangement of the third stage of rotation of the mid-gut loop, that is, the stage characterized by the fixation of certain portions of the intestine to the posterior abdominal wall, by fusion of their mesenteries with the posterior parietal peritoneum. Instead of the usual wide, obliquely placed root of the mesentery which occurs normally, there was only a narrow pedicle. This condition predisposes to volvulus of the whole of the small intestine and is a not infrequent cause of vomiting in young infants, which usually commences a few days after birth. Although no volvulus was detected in this case, it is very probable that there was an early twist which was accidentally undone when the bowel was delivered. These cases differ from pyloric stenosis in the earlier onset of the vomiting and in the presence of bile in the vomitus.

DETACHED RETINA WITH COMPLICATIONS.

By W. Lockhart Gibson, M.B., Ch.M., F.R.A.C.S.,
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A man, aged fifty years, seen in April, 1934, had been blind in the right eye for years from complete detachment of the retina. Seven days previously, following a sudden jar to the body, vision of the left eye had been reduced to seeing nothing more than shadows; it had reached this stage half an hour after the jar. There were a dense opacity of the central part of the cornea, fourteen diopters of myopia and an extensive detachment of the retina. He was put into hospital. Dr. E. J. McGuinness kindly saw the patient with me and we both searched several times for a hole in the retina or a separation of the *ora serata*; owing to the central nebula, the retina was not clearly seen and we found neither. Vision did not improve with rest in bed. Hand movement at eighteen inches still appeared as shadows. We decided that something should be attempted. Using a fine cautery, I made three punctures twelve millimetres from the limbus in the upper temporal quadrant, where the separation was the greatest. He rapidly improved. On a visit to Brisbane eight months later we were able to confirm that he had a full field of vision and saw $^{6}/_{60}$.

If a hole can be found in a detached retina, even if the detachment is very extensive, a large proportion of cures can be expected, but this case is reported with the hope that others may be encouraged to attempt treatment in such apparently hopeless cases.

Reviews.

A BOOK FOR THE TUBERCULOUS PATIENT.

Dr. Lawrason Brown, in revising the sixth edition of his book, entitled "Rules for the Recovery from Pulmonary Tuberculosis", has to some extent rewritten many parts, while including new material.[1] The value and necessity of rest, good food and fresh air are dealt with in the opening chapters and are explained along scientific and physiological lines, which should appeal to those with rudimentary knowledge of these sciences, and even to those not so well informed.

Considerable trouble has been taken to include all the recognized arguments proving the necessity of rest, and full advice is given as to how it may be carried out. After a consideration of the general constitution of foods, diet scales are mentioned for all types of cases.

[1] "Rules for Recovery from Pulmonary Tuberculosis: A Layman's Handbook of Treatment", by L. Brown, M.D.; Sixth Edition; 1934. Philadelphia: Lea and Febiger. Crown 8vo., pp. 275. Price: $1.75 net.

Little of fresh importance has been added regarding the value of fresh air, but many ways in which a patient can live in the fresh air are mentioned, with a warning that protection must be provided against severe conditions.

Exercise is considered of very late importance, and strong warning is given against taking exercise before being fully prepared for it, which is wise if the patient has to decide this question for himself.

While climate is not considered of much importance, the value of frequent currents of cold air and their tonic action on the skin are fully recognized. The importance of the pulse and temperature as a guide are stressed, while the latter third of the book deals with matters of general interest.

As tuberculosis is probably of most interest to those suffering from the disease, and as these unfortunates have seldom a true idea of all that their disease implies, the author has attempted to get down to their level of outlook and deals with each difficulty in detail in a way that the most ignorant can understand. The book should be of great assistance to all suffering from this disease.

THE WEATHER AND THE MENTAL PATIENT.

IN Volume III of a series entitled "The Patient and the Weather" the authors discuss the effects of climatic and meteorological changes on mental and nervous diseases.[1] The reader may be pardoned if he views this work with prejudice, for in the first paragraph the authors state that the general physician leaves the field of psychiatry "to a specialist who ignores the somatic background, the general, biological and medical phases in his consideration, or worse—certainly for the patient—to physicians who have by necessity turned clerks and custodians in the great public hospitals for the insane".

The general thesis of the authors is that psychic alterations may actually be initiated by meteorological events, and through the study of chemical actions they claim to demonstrate that meteorological conditions, even early in embryonic life, may have some bearing on the future stability or instability of the organism. More precisely, the authors have studied the alternation of two phases, termed "A. (anabolism-alkalinity), R. (preponderance of reduction), S. (spasm)", a condition in which there is a rise in systolic blood pressure, possibly associated with preponderance of adrenal activity, and anoxæmia with accumulation of carbon dioxide resulting from vascular spasm. The opposite phase is "C. (catabolism), O. (oxidation), D. (dilatation of the vessels)" associated with falling diastolic blood pressure and increased activity of the thyreoid. During this phase there is an alkaline side in the tissues and metabolism is diminished. The authors claim to have demonstrated these phases through their biochemical estimations, which are recorded in comprehensive graphs correlating over weeks or months as many as twenty-three different daily observations, including wind, sunshine, temperature, barometric pressure, dermography, blood pressure, basal metabolic rate, non-protein nitrogen, chlorides, cholesterol, sugar, phosphates, carbon dioxide, hydrogen ion concentration, potassium-calcium ratio, total protein, permeability, blister time, weight and psychic status. The following is a typical case summary:

> In this subject passing cyclonic waves leave distinct impressions on the behaviour. While the change is not great the record indicates that periods of greater activity and irritability or of activity and depression are associated with periods of stimulation (COD phase) that follows in the wake of the period of spasm (ARS phase). During the period of relative pressor spasm the patient seems more amiable. The status of relative acidity and lowered K/ca ratio are therefore unfavourable. The chemical and psychic picture is therefore allied to that discussed in the first cases of the series, who also had low pressures.
>
> Transposed to seasonal trends we note that increased permeability, lowered K/Ca ratio and pH and the lowered pressure of the spring is associated with more frequent periods of disturbance. Higher pH, blood pressure and K/Ca ratio, and lowered permeability are in general associated with more normal status or at least a more amiable mood. The spring then involves greater activity, the summer will therefore be more favourable.
>
> Clinically and chemically we apparently deal less often with the type of irritability that we find in manic-depressive insanity but rather with the effect of general somatic irritability incidental to the acid phase. The leptosome habitus would be in line with this.
>
> The chemical trends in general coincide with those observed in other patients during the same period of observation.

We are told that Florida and North and South Carolina offer environmental benignancy; that they are ideal regions for those who desire protoplasmic stability (presumably except when Miami is visited by a cyclone).

As regards neurological conditions, the authors have selected multiple sclerosis, tabes and paresis, poliomyelitis, and epidemic meningitis, in which it is maintained that vasomotor spasm initiated through meteorological changes plays an important part in symptomatology, if not in actual causation. Multiple sclerosis is said to occur in the United States with greatest frequency in the region of the storm tracks.

The book contains some two hundred complicated graphs and also illustrations of bodily types. It is printed in mimeograph form.

One must pay a tribute to the diligence of Dr. Petersen and his assistant, who have produced a monumental work—but what a monument!

[1] "The Patient and the Weather", by W. F. Petersen, M.D., with the assistance of M. E. Milliken, S.M.; Volume III: Mental and Nervous Diseases; 1934. Michigan: Edwards Brothers. Demy 4to., pp. 391, with illustrations.

ANATOMY AND PHYSIOLOGY FOR SURGEONS.

THE title of Lake and Marshall's book, "Surgical Anatomy and Physiology", is in itself an unusual and arresting one.[1] While the authors disclaim any attempt at a complete survey of the physiological problems of surgery, they have succeeded in developing the relationship very skilfully on suitable occasions.

The work is divided into two parts, in the first of which, the general section, the various systems—vascular, nervous, muscular, osseous, alimentary *et cetera*—are dealt with from aspects other than that of pure surgical anatomy. The regional section follows. In each of the 45 chapters of which this section is made up, the sequence, whenever applicable, is of the development, anatomy, structure, physiology, pathology and operative and practical applications. The study of the development of a region, often so essential to a clear understanding of defects and pathological tendencies, has been set forth very clearly and concisely. It has been found necessary to practise a measure of condensation to prevent the descriptive anatomy becoming too unwieldy. Even the slight discomforture which this form may engender fails to detract from the pleasure and profit which follow the reading of the book in its entirety. Few surgical teachers or practising surgeons will fail to find some fresh viewpoint or suggestive explanation, and the book may be as confidently recommended to them as to the student preparing for his examinations.

The authors are to be congratulated upon their success in infusing far more than the usual interest into the subject of surgical anatomy, upon which they have brought to bear the most recent views. It is evident, moreover, from a study of those sections dealing with operative and practical applications, that the authors speak with the confidence of a wide experience and matured judgement.

[1] "Surgical Anatomy and Physiology", by N. C. Lake, M.D., M.S., D.Sc., F.R.C.S., and C. J. Marshall, M.D., M.S., F.R.C.S.; 1934. London: H. K. Lewis. Demy 8vo., pp. 898, with 238 illustrations. Price: 30s. net.

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THE DIAGNOSIS OF BRAIN TUMOURS.

The papers by Dr. Rupert M. Downes and Dr. Ellis Murphy, dealing with the diagnosis of cerebral tumours, and published respectively in the issues of January 26 and February 2, 1935, are of such importance that they should not be allowed to pass without comment. Cerebral tumours are stated by Ewing to be responsible for probably 1% of all deaths. Ewing refers to the finding by Bruns of 210 brain tumours among 11,500 neurological patients, but adds that Hirsch saw only 14 among 8,000 nervous cases. Among 14,005 autopsies at Munich and Heidelberg 135 brain tumours were recorded by Seydel and Beck. Cleland reported in this journal several years ago that in a series of 1,000 autopsies performed at Adelaide Hospital 16 brain tumours were found. The occurrence of tumours of the brain is therefore by no means rare; and it is not surprising that the enormous progress of recent years in cerebral surgery and the perfection of newer diagnostic methods have focused attention on them and have emphasized the need for early diagnosis. The discovery of the sufferer from a cerebral newgrowth, so that he may be placed under the care of a neurological surgeon, falls to the lot of almost every medical practitioner, though the removal of tumours from the brain and the use of such diagnostic procedures as ventriculography and arteriography are and will remain the province of the few.

Dr. Murphy has given an excellent description of the symptoms usually associated with brain tumours. The triad, headache, vomiting and papillœdema, is everywhere regarded as pathognomonic of intracerebral newgrowth. When these symptoms are present, the veriest tiro will probably make a correct diagnosis. But they are not always present, either singly or in combination; and in these circumstances other indications have to be sought. Dr. Murphy has referred to certain vague initial symptoms, and Dr. Downes has quoted the view of Bailey that the only cardinal symptom is a steadily increasing afebrile alteration in nervous function. While asking our readers to consider the remarks of Dr. Murphy and Dr. Downes, we would also draw attention to a discussion that took place at the annual meeting of the American Medical Association last year, when the absence of classical symptoms in cases of brain tumour was discussed by A. E. Bennett and J. J. Keegan.[1] These two authors pointed out that the classical symptoms of increased intracerebral pressure are absent from 15% to 30% of large groups of patients with intracerebral tumours studied by different observers. They give short histories of twelve patients suffering from tumour who manifested no symptoms of generalized increased intracranial pressure. In seven of these cases a diagnosis was made by encephalography. The histories make most interesting reading and should be studied by all who have access to the journal in which they appear. The initial symptoms of one patient were attacks of numbness of the right foot and leg, transient ataxia and momentary blurring of consciousness. Another patient complained of cramping of the right foot, and this was followed by clonic spasm of the right half of the body. In another instance what was thought to be a traumatic hæmatoma was found

[1] *The Journal of the American Medical Association*, January 5, 1935.

to be an oligodendroglioma. In another case the clinical picture suggested an encephalitic infection. One patient was treated for ten years for epilepsy. And so on. Cases such as these show the need for watchfulness. Bennett and Keegan point out that the location of a tumour and not its size is responsible for the degree of hypertension present. They stress the importance of local pressure signs and show that these signs may in certain cases clinch the diagnosis before generalized pressure signs appear.

Ventriculography, it will be stated, may reveal tumours when increased pressure signs do not make the diagnosis obvious. In many cases it will. Dr. Downes, however, has emphasized the fact that ventriculography is not without risk; and it is important to remember that it or similar complicated methods of examination should not be used when clinical methods are sufficient to establish a diagnosis. Further, when it is used, the surgeon must be prepared to go on at once, if necessary, with the operation of craniotomy. Only the expert can determine which patients should be submitted to special methods of examination. The main problem lies in discovering which patients may reasonably be suspected of harbouring an intracranial growth. Dr. Downes has put his finger on the most important consideration when he writes that the chief step in diagnosis is that the possibility of the existence of an intracerebral tumour should occur to the medical attendant. If the medical attendant keeps this possibility in mind, and if he remembers that intracranial tumours do not always produce classical symptoms, patients will be brought to operation earlier and more lives will be saved.

Current Comment.

THE VITAMIN AND IODINE CONTENT OF COD LIVER OIL.

Since time immemorial cod liver oil has been used with benefit in many diseased conditions, especially rickets and tuberculosis. Much speculation has been made as to the reason for its efficacy. But Cushny's "Pharmacology and Therapeutics" (1918) tells us that "it has not been shown to have any action apart from that of an easily digested food, and its superiority to some other fats and oils has not been satisfactorily established". A. J. Clark's "Applied Pharmacology" (1923) states that "the first theory advanced was that the oil contained a higher proportion of unsaturated fatty acids than did other oils and, therefore, was absorbed more readily". With the discovery of vitamins in the oil its beneficial effects were attributed to such constituents, especially in rickets. But evidence is accumulating that in keratomalacia, xerophthalmia and rickets, due to deficiencies of vitamins *A* and *D*, the oil may effect cure when carotene (the precursor of vitamin *A*) and irradiated ergosterol, supplying vitamin *D*, have failed. Before vitamin *D* had been differentiated from vitamin *A* it was considered that the cod derived its vitamin eventually from the plankton (chlorophyll-containing diatoms and green algæ) in the upper ocean waters, the vitamins being synthesized by sunlight in such organisms. Plankton is eaten by small fish that become food for the cod and halibut, which store the vitamins largely in the liver. But later observations show that plankton is not a rich source of vitamin *D*, although it contains vitamin *A* in abundance. The cod is a "ground" fish subsisting partly on invertebrates found at the bottom of the ocean. According to Bills, its principal food about Newfoundland is the caplin, which does not contain enough vitamin *D* to explain its abundance in the cod during its summer fattening. Possibly the cod synthesizes vitamin *D*. S. W. Clausen has said that, if rapid storage of vitamin *A* be required, halibut and cod liver oil would seem better than carotene, as this is less rapidly absorbed than vitamin *A*. Carotene in conditions of fever or diarrhœa is badly absorbed. H. Steenbock and his colleagues stated that vitamin *D*, produced by irradiating ergosterol by a quartz mercury vapour lamp, is not the same body as that found in cod liver oil. Possibly the vitamins of cod liver oil are in some subtle way different from the standard *A* and *D*; or perhaps some other substance exists in the oil, giving it additional virtues.

Hupper de l'Orme first detected iodine in cod liver oil in 1836. In Hare's "Practical Therapeutics" (1925) it is written that "cod liver oil depends on a number of substances for its peculiar effect. The iodine certainly exerts definite alterative powers". Cushny's "Pharmacology and Therapeutics" states that in cod liver oil iodine and bromine are present in traces and in very much smaller amount than is generally believed. The usual statement is that 0·03% to 0·04% of iodine exists in the oil, but some oils have been found to contain only one-hundredth of this amount. The British Pharmacopœia stipulates an iodine value of 155 to 173.

A. D. Holmes and R. E. Remington have estimated the iodine content of American cod liver oils.[1] Remington and others had previously reported that the iodine content of the same agricultural product, grown in the same locality, varied by 200% to 300%.

[1] *American Journal of Diseases of Children*, January, 1935.

Sea products are richer in iodine than those of the land. The iodine content of sea water is more constant than of the soil, but large variations in the iodine content of oysters from different parts of the United States coast have been reported. T. von Fellenberg found 5,100 and 5,620 parts of iodine per billion in two samples of cod liver oil. G. Lunde, in oils from Norway, found 15,200 parts per billion in cold cleared oil, 14,200 in crude medicinal oils, and 9,900 in pale medicinal oils. He was of opinion that the iodine content of fish depended on the location of the fishing grounds. Remington, in two samples of medicinal grade oils, found an iodine content of 10,500 and 10,400 parts per billion. His investigations included fruit and vegetables grown on South Carolina soil rich in iodine. He found that cod liver oil contained vastly greater amounts of iodine than commonly used food materials. Holmes and Remington analysed twenty samples of crude medicinal oil (not destearinized) from the North Atlantic coast of the United States, Nova Scotia, Quebec and Newfoundland. The range of values was from 3,590 to 14,940 parts per billion, with an average of 8,640. The iodine content varied with the locality. Nova Scotia had 13,260, Massachusetts 4,930, and Maine 3,590. These differences cannot be explained. The oil is produced on the shores of Massachusetts in the winter and on the northern areas in summer. The cod migrates extensively. It prefers water of a temperature between 1·6° and 10° C. The pursuit of food entails migration, and breeding migrations are due to a search for shoal water in which to spawn. Possibly oil comes from the same school of fish at northern points in the summer and southern points in the winter.

The iodine content of sea water was first determined by L. W. Winkler (1916). He found 38 gammas per litre in the Adriatic. Subsequent analyses have shown 18 to 23 gammas. This may be increased locally by liberation of iodine from decaying vegetation. It is improbable that the differences in iodine content of cod liver oils are influenced by the amount which the cod derives directly from sea water. The amount of iodine in the flesh and intestinal contents of the cod is much greater than in surface fish. Crustaceans and shellfish contain more iodine than true fish. Possibly the forms which are food for the cod are more numerous in certain areas, thus causing the difference in the iodine content of various oils. It has been proved that the iodine of cod liver oil is absorbed and passes into the milk of lactating cows. T. von Fellenberg found better absorption and retention of iodine from cod liver oil than from iodides, watercress or sardines; but he considered urinary elimination only. Others have shown that in women urinary excretion of iodine is highest in the premenstrual and early menstrual periods. Only a portion of the iodine absorbed is excreted in the urine. Elimination occurs also by the sweat, saliva, tears, milk, sebum and nasal mucus. G. M. Curtis found that the iodine of the blood was increased to the same degree by the use of cod liver oil and of iodized salt.

We are left with the knowledge that cod liver oil possesses some therapeutic attributes of great utility that are not yet adequately explained.

SPIDER BITE.

Poisonous spiders have aroused more than a passing interest in Australia, and whether the more pretentious term of "arachnidism" or its simpler synonym is used, the subject will always awaken very definite clinical memories in those who have witnessed the effects of bites from the *Atrax* or the *Latrodectus*. It might be thought that after all the work that has been done by Bogen and others in America, and by Ingram and Musgrave and by Kellaway in Australia, there could be no doubt about the danger attendant on attacks by these venomous spiders, but it is apparent from a perusal of the literature that the evidence is regarded as indirect by some writers. It is pointed out that the results of animal experiments are not consistent and that several observers have claimed to have been bitten without bad effect. These arguments are, of course, quite valueless, for the susceptibilities of laboratory animals to toxins and venoms of different sorts vary greatly and bear no necessary relation to the size and strength of the animals. Even in the same animal, human or otherwise, there is a considerable variation in the response to poisons of various kinds. Moreover, it has not been proved that the venom of spiders is constant in the species or at different seasons of the year. But if anyone is still sceptical of the urgency of the symptoms that may follow the bite of *Latrodectus*, he may be impressed by the account of an experimental bite in man.

A. W. Blair allowed a "black widow" spider (*Latrodectus mactans*) to bite him and has recorded the exact details of his experience and his subsequent illness.[1] Blair divides the symptoms into three stages: those of lymphatic absorption, those of vascular dissemination, and those of elimination. The first stage was manifested by local pain in the finger, followed by swelling and some sweating round the site of the bite. Definite pain in the cubital lymph gland was also felt, and this spread to the axilla and then to the side and front of the chest. Within an hour the stage of vascular spread began, the blood pressure falling and abdominal pain becoming evident. This pain became very severe and the muscles later were rigid; within two hours from the biting the abdomen was board-like and the pains in the lumbar and abdominal regions were agonizing. One of the experimenter's colleagues who was called to see him two and a half hours from the onset, stated that he could not recall having seen more abject pain manifested in any other medical or surgical condition, and that there were all the evidences of profound shock. Half an

[1] *Archives of Internal Medicine*, December, 1934.

hour later the systolic blood pressure was only 80 millimetres of mercury and the diastolic 50, and the pulse rate was 120. At this time also the leucocyte count was estimated to be 13,000, whereas at the beginning of the experiment it was 8,000. Other symptoms were laboured respiration and muscular spasms in the limbs. Sweating is mentioned as occurring, but more particularly in the last stage, that of elimination, and this symptom was also one of the commoner of those found by Bogen in his analysis of 15 cases. It may be remarked that it is a common and outstanding symptom in the cases of bite by the *Latrodectus hasseltii*. As other observers have reported, pain was one of the most severe and troublesome of all the symptoms. In his comment Blair says that no mention is made in the literature of a period of severe shock during the time when the agonizing pains are felt. This is surprising, for shock has been specially mentioned in the reports of Australian cases, and Ingram and Musgrave advise adrenaline and pituitrin to combat shock. Blair regards the second stage as the most urgent, with which all will agree, and, like other writers, he refers to the ease with which the condition could be mistaken for an acute abdominal emergency. He also mentions another interesting point, that is, that the stage of elimination is characterized by hypertension, diaphoresis, slight rise in temperature, leucocytosis and urinary evidence of renal damage. This picture, together with the slight œdema of the face and extremities seen in some cases, suggests a transient toxic nephritis. The serum of convalescents has been used in the treatment of this type of poisoning, and Blair remarks that the development of any degree of immunity during convalescence is really unproved. In the present instance he had an opportunity of repeating his experiment in order to add to the knowledge on the subject, but wisely refrained. Such an experiment is an ordeal that few would care to face once, without contemplating a repetition, even though perhaps protected by a problematic immunity.

This courageous study is of value; if there are any to be convinced, they should now agree that these spiders are highly toxic to man and that their bite may be very dangerous. All Australian practitioners should be conversant with the symptoms of poisonous spider bite, for not only are the patients often very ill, but they occasionally die, and, in addition, the diagnosis may be obscure where there is no clear history of an actual bite.

THE ÆTIOLOGY OF APPENDICITIS.

There is a general impression that appendicitis has increased both in frequency and severity. To put this on a statistical basis is difficult, for the available records of hospitals are not always easy to interpret on such a subject when the period of inquiry goes back to the days of less energetic surgical treatment, and therefore of less accurate knowledge of the pathological conditions present. Moreover, as most of those will agree who have essayed an analysis of hospital histories, these records are only too often incomplete and lacking in reliability. W. H. Bowen, in a study of the ætiology of appendicitis, states that he believes the records of Guy's Hospital, which he consulted in the compilation of his paper, to be accurate and complete, being well controlled by the supervision of the registrars.[1] He is to be envied in having access to such admirable material, and the staffs of hospitals should remember the part they may play in medical research by the faithful performance of their duty in keeping full and correct records. Another difficulty in such a study is the subdivision of the cases into different categories, especially the mild and the severe. Bowen has regarded as mild those showing no evidence of infection by the colon bacillus in the peritoneal cavity, the severe cases being those with abscess formation or general peritonitis. This classification is open to the objection that a local plastic peritonitis, which would allow the placing of a case in the mild group, might be associated with local gangrene and thus be potentially severe. However, the author remarks that these are rare and that the area of gangrene is usually superficial, not penetrating the coats of the appendix.

In order to arrive at some idea of the degree of severity of the inflammatory process he has examined 150 appendices removed during one year from patients suffering from acute attacks in the Addenbrooke's hospital. Of these, 69 were only mildly inflamed, though eight of these showed some localized gangrene of the mucosa, and 81 were of the severe order. Of the latter, 55 showed extension of the process of gangrene to the peritoneal coat of the organ. The careful pathological survey of the material from these cases, together with consideration of the histories, has been used to form some estimate of the importance of the possible ætiological factors. In the consideration of these the questions of constipation and purgation have come up. Bowen finds no support for the idea that appendicitis has any relation to constipation. That the severity of the process may be seriously increased by the use of purgatives and large enemata is beyond doubt, and this fact is stressed by him. He further draws attention to the success attending the methods involving physiological rest, as practised in the pre-surgical days, when the influence of Hilton, famous for his lectures, "Rest and Pain", no doubt dominated Guy's Hospital. The value of rest in appropriate cases has been the subject of communications to the pages of this journal. There is no doubt that the firmly established domestic custom of administering an aperient to children and others suffering from abdominal pain has hastened the exit from life of not a few of the victims of what is a dangerous practice, and it is a pity that this piece of knowledge is not more widely disseminated among the laity.

[1] *Guy's Hospital Reports*, October, 1934.

On examining the other possible factors in the causation of the attacks in the persons operated upon, Bowen does not attribute any degree of importance to the presence of bands causing kinks in the appendix. He believes that many of the firm bands found are congenital and that they seldom cause true obstruction of the lumen of the appendix. In fact he states that he has more frequently seen stagnation in the appendix when no such bands were present. Vulnerability of the blood supply to the appendix has also been cited as an important ætiological factor, but it would appear that the blood supply is not only very constant, but also rich and amply supplied with collaterals. Threadworms have been regarded as possibly important by some, but Bowen found them in only 1% of cases. He further quotes the experience of Fischer that appendicitis is very rare in China, although intestinal parasites are extremely common. Australian surgeons might easily produce a greater percentage than one, though probably general impressions are a risky foundation for an opinion, but they would be likely to agree that the presence of threadworms *per se* is not a factor of importance in the causation of appendicitis.

Further discussion of the possible causes leads us into the realm of hypothesis, where it is hard to adduce positive proof. Bowen does not think that an enterogenous infection is a likely cause, and remarks that, were this so, more young persons should suffer from appendicitis, since enteritis and colitis are common amongst them. With regard to the theory that the disease is blood spread, he is likewise sceptical, looking with some doubt on the conclusions of Rosenow, and pointing out that an antecedent focus is absent in the majority of cases, as far as can be found. He refers to the suggestion of Lansdown and Williamson that all inflammatory lesions of the gastro-intestinal tract may be all included in one pathological concept, inflammation of the solitary lymphoid follicles. These excursions into speculation cannot be pursued here, nor those upon the possible intrinsic susceptibility of the appendix to inflammation, but the importance of the stercolith should be pointed out, for this concretion, whatever may be its exact method of formation, is of mechanical importance in determining the severity of the disease. Bowen believes that acute appendicitis begins as an infective catarrh. This vague statement of a pathological creed must be taken as an indication of the need for further work on this common and serious disease.

THE TRANSMISSION OF SYPHILIS BY BLOOD TRANSFUSION.

When a transfusion of blood is given in the course of medical treatment, every care should be, and generally is, taken that the donor is free from disease that may be communicated to the recipient. Syphilis is the condition that will be thought of at once in this connexion. The literature has many accounts of syphilitic infection transmitted by blood transfusion, and it would be safe to conclude that transmissions of this kind occur that are not reported. The matter is one of considerable moment and attention should be drawn to a recent article published by H. W. Jones, T. K. Rathmell and C. Wagner.[1]

These authors state that the use of controlled serological tests will in most cases prevent the possibility of the transmission of syphilis. At the same time they point out that transmission may occur in emergency, in the absence of adequate laboratory facilities, as a result of lack of intelligence on the part of familial donors, because of purposive untruthfulness, or when a transfusion is given from a donor who is in the early stage of the disease. Many medical practitioners will say at once that if the donor is subjected to a Wassermann or Kahn test, or both, and does not yield a reaction, there will be no danger. In the majority of cases this would be true. But sight should never be lost of the fact that an absence of response to the Wassermann or similar test does not necessarily denote the absence of syphilitic infection in the person tested. In 1930 Tzanck and Werth reported the history of a professional female donor who had given blood eighteen times in the years 1925 and 1926, and whose blood had been tested six times in three years. Suddenly, at the age of forty-six, she became affected by hemiparesis and by icterus, and her response to the Wassermann test became positive. It is immaterial to the present discussion that, so far as is known, none of her eighteen recipients became affected by syphilis.

Jones and his fellow workers have discovered in the literature twenty-one cases in which syphilis has been transferred either from the donor to the recipient or *vice versa*. They add four cases in which donors were found to be syphilitic; in one instance the disease was transmitted, in three it was not. The patient who became infected received his transfusion on account of pernicious anæmia. He manifested a rash and had convulsive seizures and psychotic symptoms. It was thought that his history suggested syphilitic meningitis or neurosyphilis; at autopsy chronic meningitis was found. In two of the other cases the donors had no knowledge of any infection.

Jones and his co-workers offer many suggestions for the avoidance of this disaster. We do not believe that the "take a chance" attitude to which they refer is at all common. It is, however, possible that undue reliance is placed on the result of one test. Of course, laboratory facilities should always be available to medical practitioners, and reports should invariably be written. Most important is the need for transfusion to be carried out by none but efficient and well trained workers. It would also be wise to follow Jones, Rathmell and Wagner in their belief that inactive syphilis may be transmitted.

[1] *American Journal of Syphilis and Neurology*, January, 1935.

Abstracts from Current Medical Literature.

DERMATOLOGY.

Necrobiosis Lipoidica Diabeticorum.

E. P. Zeisler and M. R. Caro (*Archives of Dermatology and Syphilology*, December, 1934) report two cases of *necrobiosis lipoidica diabeticorum* (Urbach-Oppenheim) with necropsy observations in one case. The condition is a rare disease of the lipoid and carbohydrate metabolism characterized by yellow to buff coloured scleroderma-like plaques on the lower extremities. The histological changes are pathognomonic and consist primarily of vascularitis with perivascular lymphocytic infiltration and extensive necrobiotic areas in the corium containing swollen collagen fibres which are infiltrated by droplets of lipoids the nature of which has not been determined. The pathogenesis of the disease is explained by a deposition in the necrobiotic intercellular connective tissue of the excess lipoids present in the blood following changes in the cutaneous blood vessels caused by the diabetes. The lesions must be differentiated from xanthoma, amyloidosis, scleroderma and similar dermatoses. The disease is chronic and resistant to treatment with insulin and to diets low in fat.

Lichen Nitidus.

M. S. Wien and M. O. Pearlstein (*Archives of Dermatology and Syphilology*, December, 1934) report a case of *lichen nitidus* in a white woman, aged fifty years. The eruption first appeared on both knees and elbows and gradually spread to involve the entire extremities. The eruption consisted of small, grouped, shiny pimples the size of a pinhead. The lesions had a yellowish waxy hue. The lesions later became diffusely disseminated over the shoulders, breasts and abdomen. The hips to the lower third of the legs presented discrete follicular lesions the colour of cayenne pepper, with minute hyperkeratotic central puncta that gave the sensation of a nutmeg grater. Physical examination gave negative results.

Scleroderma, Acrosclerosis and some Kindred Diseases.

J. Sellei (*The British Journal of Dermatology and Syphilis*, December, 1934) differentiates the conditions of scleroderma in its various forms and acrosclerosis, and mentions Morvan's disease, syringomyelia, Bürger's disease and Raynaud's disease in a differential diagnosis. The treatment suggested in scleroderma is: (*a*) Raw pancreas, 100 to 200 grammes every day at breakfast in warm (not hot) bouillon or mixed with potato purée. (*b*) Pancreas ferments in the form of tablets containing amylase, lipase and trypsin (pancrin tablets), to be taken daily on an empty stomach, three to four an hour before breakfast, and two before the other two meals. (*c*) Ferments containing duodenum in tablet form, together with pancrin tablets, two tablets twice a day. (*d*) Liver extracts together with pancreas treatment. The functional disturbance of the pancreas can be established from the positive reaction in the atoxyl lipase resistance test, which gives no reaction in acrosclerosis. Treatment suggested in acrosclerosis is by acetylcholin, padutin and kindred drugs, muscle and skin extracts, histamine iontophoresis, salves and parathyreoid extract.

Verrucose Pyodermia.

W. M. Goldsmith (*The Proceedings of the Royal Society of Medicine*, February, 1935) reports a case of verrucose pyodermia. The patient was a male, aged forty-six years. The whole of the dorsum of his left hand was occupied by a scar. Along its upper border there was an interrupted, serpiginous, raised, warty wall containing fistulous tracts along which pus escaped. The peripheral edge was bluish and smooth. Somewhat similar but less pronounced lesions were present on the right hand, left forearm and right arm. The history was that nine months previously a splinter of Oregon pine, "filthy and alive with creatures", entered the back of the patient's left hand. A swelling appeared which discharged pus and gradually extended. The Wassermann test gave no reaction. A moderately positive reaction followed the intracutaneous injection of one in 5,000 tuberculin. No blastomyces or other fungi were found in smears. On culture *Staphylococcus albus* and a proteolytic bacillus, probably *Bacillus proteus*, were grown. In reporting the case, the author pointed out that Ferguson Smith had stated that the condition was due to *Staphylococcus aureus*, together with allergy. In this case no *Staphylococcus aureus* was present. It was by no means certain that the streptococcus, if responsible for the abscess, was also responsible for the original papillomatous lesions.

Aldo Castellani, in discussing the case, said that the history was in favour of a diagnosis of blastomycosis; so was the verrucoid appearance and the clearing of the lesions in the centre, while they spread in the periphery. He thought that the condition was pseudo-blastomycosis or *dermatitis vegetans*.

Henry MacCormac said that he thought that many different organisms could produce the reaction seen in this patient. He had seen a similar case that was regarded as being tuberculous.

Granuloma Annulare.

F. H. Grauer (*Archives of Dermatology and Syphilology*, December, 1934) reports a case of *granuloma annulare* affecting the subcutaneous tissue of the scalp. Although the association of subcutaneous nodules with typical cutaneous lesions of *granuloma annulare* is suggestive of their ætiology, microscopic examination is necessary to establish their identity. There was no history of rheumatism or tuberculosis. Tuberculin reactions were positive only with concentrated material, namely, with 0·1 cubic centimetre of undiluted old tuberculin and one milligramme of tuberculin protein (tuberculin precipitation test). No evidence of tuberculosis could be found by thorough clinical and X ray investigation.

UROLOGY.

Vesico-Vaginal Fistula.

D. Fonseca (*Journal d'Urologie*, August, 1934) proposes the use of distension of the bladder in order to open up the folds of the vesico-vaginal fistula by the vaginal route. This distension is effected by introducing a child's toy balloon into the bladder through the urethra and then distending it with water. Not only is the field of operation better exposed by this ingenious plan, but, in addition, the surgical manœuvres are facilitated by the fact that the edges of the fistula rest on a supporting bed.

Diagnosis of Cortical Suppuration of the Kidney.

Whenever doubt exists as to the presence of perinephric abscess, perinephritis, renal carbuncle or cortical abscess of the kidney, C. P. Mathé (*Urologic and Cutaneous Review*, September, 1934) recommends pyelography in the vertical, horizontal and Trendelenburg positions. He states that, in the presence of clinical signs, immobility of the kidney as demonstrated by these pyelograms is pathognomonic of perinephric adhesions, and the diagnosis may be confirmed in the presuppurative stage.

Epispadias in Women.

O. Mercier (*The British Journal of Urology*, December, 1934) describes the technique used in a successful operation for epispadias in a woman. The condition is a rare one and is seen by the surgeon only when incontinence is present in some degree. The author briefly reviews operations previously practised and states that he has borrowed from each of them. It is noted that unless a sphincter is provided by transplantation of muscle, continence can be achieved only by the use of an inflatable rubber bag. The author's technique aimed at lengthening and narrowing the urethra, providing a sphincter and reforming the labia and clitoris. A flap from the anterior vaginal wall was used to create a longer urethra. Two stumps of tissue were found, one on either side of the short urethra, and were believed to be rudiments of the *sphincter urethralis*. These were

united in the mid-line as in the operation for radical cure of cystocele. The operation concluded with restoration of the clitoris and labia. Suprapubic cystostomy was used for bladder drainage for fourteen days. Thereafter the patient became completely continent and has remained so for three years.

Malakoplakia Vesicæ.

A. E. CHISHOLM AND G. R. TUDHOPE (*Edinburgh Medical Journal*, November, 1934) describe a case of malakoplakia of the urinary bladder. The condition is rare, only two cases being recorded previously in Great Britain. The patient, a female, about sixty-one years of age, complained of vaginal pain, incontinence of urine and loss of weight. A tumour was palpable above the pubis and a sound passed into the bladder lay behind the swelling. No cystoscopic or cystographic studies were made, but the tumour was excised. The wound healed, but incontinence continued and the patient subsequently became mentally deranged. The characteristic feature of *malakoplakia vesicæ* is the presence on the inner surface of the bladder of rounded or oval plaques which stand out from the mucosa. They are usually pale, often yellowish in appearance, and they are frequently umbilicated. Microscopically the plaques are formed of granulation tissue in which there are masses of peculiar large polygonal mononuclear cells lying in a delicate areola stroma. The epithelium may be destroyed in parts. The large cells (malakoplakia cells of Van Hausemann) contain vacuoles, homogeneous refractile spherical bodies (Michaelis-Gutmann bodies) frequently showing concentric lamination.

Congenital Hypertrophy of the Bladder Neck.

P. CAUSA (*Journal d'Urologie*, August, 1934) draws attention to the clinical entity described by Marion in 1927 as a congenital hypertrophy of the vesical internal sphincter muscle. He describes a personal case presenting a peculiar and remarkable symptomatology. This boy of fifteen passed about seven litres of urine each day and suffered from thirst. On this account he had been treated as a diabetic, though the sugar had actually been determined. A large abdominal tumour was found, and this proved to be a chronically distended bladder containing about 2·5 litres of residual urine. In the absence of visually determinable lesions (by X rays, cystoscopy *et cetera*) the diagnosis of congenital hypertrophy of the internal sphincter was made, and this muscle was excised by the suprapubic cystotomy method of Marion, in which a circular incision through the mucosa of the bladder neck is made. At operation the internal meatus was found to be stenosed. Microscopy determined an excessive hypertrophy of the internal sphincter muscle. The plain muscle bundle interlaced freely, running in all directions; the muscle cells were larger than normal, and their nuclei showed increased chromatophilia. Recovery was slow on account of renal damage, but after seven months all vesical residue had disappeared and the polyuria had decreased by two-thirds; in addition, the general condition was distinctly improved. This disease is somewhat rare, but, on the other hand, it is possibly often not diagnosed. Other types of bladder neck stenosis (not to be confused with true prostatic hypertrophy) have been described by Marion and by Legueu as being due to hypertrophy of local glandular elements (for example, median vesical bar) or of local connective tissue elements (for example, fibrosis resulting from prostatis), but this entity is a true congenital hypertrophy of the sphincter muscle.

Cutaneous Ureterostomy in Vesical Tuberculosis.

J. FRESNAIS (*Journal d'Urologie*, October, 1934) considers that exclusion of the bladder is definitely indicated in rebellious cystitides following nephrectomy for tuberculosis, when (*a*) the intensity of the pain in spite of all treatment renders life intolerable, and (*b*) when the existence of ureteric reflux threatens tuberculous infection of the remaining kidney. Cutaneous ureterostomy in the iliac portion of the anterior abdominal wall gives the best results and, whatever the final result, the immediate object of relieving the patient is always achieved. A rubber catheter is kept permanently in the ureter. It is easily removed by the patient for cleansing and sterilizing purposes and, moreover, the patient can inject mild antiseptics into the kidney whenever required.

Aberrant Renal Vessels as a Cause of Hydronephrosis.

G. PETREN (*Zeitschrift für Urologie*, March, 1934) inquires into the extent to which aberrant renal vessels cause hydronephrosis. Aberrant vessels actually occur in about 20% of all cases, but the question of abnormal renal vessels is of more interest in the pathological and clinical domain than in the anatomical. About one-third of all cases of hydronephrosis or pyonephrosis, not of congenital or stone occlusion origin, are due to renal mobility, and very often the blockage is due to a combination of this mobility with the presence of rigid abnormal vessels. Recently several workers have tried to prove that all hydronephrosis arises originally from a single factor, for example, renal mobility according to Marion, or achalasia of the uretero-pelvic junction according to Legueu. The author, however, considers that the pathogenesis can hardly be the same for all cases. The actual cause can best be determined by pyelographic, pyeloscopic and operation observation. The question of pathogenesis can be further elucidated by remote pyelographic, pyeloscopic and functional studies in all cases in which conservative procedures (denervation, vessel division *et cetera*) have been performed. In regard to treatment, so long as the question of the ureteral pathogenesis remains obscure or in dispute, the operative therapy has no certain basis. In the last two decades urologists have striven more and more to perform conservative operations, that is, to avoid nephrectomy, whenever possible. We must always inquire, in the face of an apparent mechanical blockage, whether the obstruction is actually mechanical, and try to be sure that it is not functional, as, for instance, in neuro-muscular disturbance.

Sclerosis of the Vesical Sphincter.

R. CHWALLA (*Zeitschrift für Urologie*, March, 1934) discusses the ætiology and treatment of sclerosis of the internal vesical sphincter muscle. It has been his experience that inflammatory processes account for only a very small proportion of all cases. The majority of cases belonged to the so-called idiopathic group, of doubtful origin. Sometimes this is simply a hypertonic condition of the sphincter muscle arising from changes in the central nervous system. In all cases the basis of cure is complete division of the sphincter muscle. The author considers that the only way to be sure of effecting proper division of the muscles and good dilatation of the internal meatus is by open suprapubic operation with a wedge or V-shaped incision. Indeed, open operation with direct palpation is often the only way in which the condition can in many cases be satisfactorily diagnosed. Another advantage is that after digital dilatation small adenomata are often palpated and should be enucleated. In his series of 70 such operations the mortality was 7%, which, when all circumstances are taken into account, the author considers very satisfactory.

Urinary Tract Anomalies.

G. MARCUCCI (*Archivio Italiano di Urologia*, August, 1934) passes in review all the types of congenital anomaly which arise from whims of development in the urinary tract. Every malformation assumes, so far as pathological changes are concerned, its particular importance in proportion to its effect on the kidney by reason of interference with the outflow of urine. Some malformations produce stasis by simple mechanical obstruction of the urinary outflow; some by disturbing the harmony of muscular action along the whole urinary tract which is necessary for unimpeded urinary outflow; other malformations are the expression of a constitutional deficiency, myogenic or neurogenic, in the pyelo-ureteral excretory system. A vicious circle is set up: stasis favours infection, infection maintains the stasis by inducing congestion and spasm. All grades of renal disease are then developed, even, in the worst cases, a functionless, pyonephrotic organ.

Special Articles on Treatment.

(Contributed by request.)

XLVIII.

TREATMENT OF RETAINED PLACENTA AND POST PARTUM HÆMORRHAGE.

The most frequent cause of both retained placenta and *post partum* hæmorrhage is the failure to appreciate properly the factors concerned in the normal separation of the placenta and the resultant improper management of the third stage of labour. It is necessary, therefore, first to describe briefly the phenomena associated with placental separation and to stress the importance of careful treatment by the obstetrician during this period.

As soon as the child is born there is usually a cessation of the uterine contractions, but it will be noticed that the uterus becomes much smaller, so that the fundus descends to about the level of the umbilicus. This is brought about, not by uterine contraction, but by uterine retraction.

The reduction in size of the uterus diminishes the area of the placental site, and the placenta, being unable to follow, tends to become separated from the uterine wall. A second effect of the retraction is to limit hæmorrhage by strangling the vessels supplying the placental site where they pass through the middle or reticulated muscular layer.

As soon as the uterine contractions recur these effects are accentuated so that the placenta is eventually separated and is then expelled into the lower uterine segment or vagina. A certain amount of hæmorrhage, forming a retro-placental hæmatoma, also aids in the placental separation.

As the placenta leaves the uterus the membranes are stripped off the uterine wall and the placenta, now being beyond the contractile sphere of the uterus, has to be expelled from the vagina either by the voluntary efforts of the patient or by pressure on the uterus by the attendant. Uterine contractions followed by further retraction occur, effectively preventing any further gross bleeding from the placental site.

The knowledge of what is happening inside the uterus while the placenta is separating is the best guide as to the correct treatment during the third stage of labour.

It is usual to follow the fundus down with a hand on the abdomen and then to place the patient on her back, for in this position the uterus is more easily watched. The cord should now be drawn gently down to its full extent. If there is reason to believe that the bladder contains an appreciable amount of urine, it should be emptied by catheter.

The hand should be held motionless just above the fundus, in which situation the aortic pulse can be watched and the onset of uterine contractions recognized. It is at this stage that the attendant should exercise all possible patience. There may be some delay before contractions recommence, but in the absence of bleeding the fundus must not be stimulated into what may be an irregular contraction.

When the placenta has been expelled into the lower uterine segment or vagina there is usually a little fresh hæmorrhage and more of the cord will have appeared, while abdominally a swelling in the hypogastrium may be evident; the fundus may rise. The uterus, when contracted, is regularly hard all over and now has changed its shape from a globular organ to one which is flattened from before backwards.

When the placenta has reached the upper part of the vagina expulsion may now be completed by the Dublin method, which consists in first anteverting the contracted uterus and then pushing it downwards and backwards and so forcing the placenta through the vagina and vulva. Care must be used to prevent tearing of the membranes. After using the Dublin method the uterus should be drawn upwards into the abdomen, where it is more easily controlled.

Retained Placenta.

Definition.—Normally the placenta becomes separated in from fifteen to thirty minutes, and a retained placenta is considered to be one which fails to be expelled from the uterus in one hour.

If there is no abnormal hæmorrhage, the treatment already described for the conduct of the third stage of labour should be persisted with, and no attempt should be made to interfere with the uterus. At the end of one hour, however, steps may be taken to deliver the placenta, though it must be remembered that the safety of the patient is best served by waiting still longer, until it becomes evident that the placenta is not going to be extruded without help.

Ætiology.—The most important predisposing cause of retained placenta is bad management of the third stage, while other ætiological factors are a full bladder, uterine inertia due to prolonged labour, continued anæsthesia or repeated doses of sedative drugs and, rarely, abnormal adhesion of the placenta. These conditions must be remembered and prevented where possible.

The immediate cause of retention of the placenta is deficient contraction and retraction of the uterine muscle. This may be general or local. With general atony the placenta becomes separated by retroplacental hæmorrhage, but is not expelled by the uterus, and profuse *post partum* hæmorrhage is the result. Where there is localized deficiency in the uterine tone the placenta becomes partially detached, while elsewhere it still remains adherent to a moderately atonic area. In this case the hæmorrhage is not usually so urgent.

Treatment.—When it is realized that the condition is one of retained placenta, a catheter should be passed, if this has not already been done, and then the fundus of the uterus should be massaged into a contraction and an attempt made to express the placenta by Credé's method.

Credé's method of expression is first to antevert the contracted uterus and then to compress it strongly between fingers and thumb, at the same time pushing it downwards and backwards in an attempt to squeeze the placenta out of the uterus. This operation usually produces considerable shock to the patient, and therefore should be used once only at this stage. If Credé's expression fails, the patient should be anæsthetized and then the method should be repeated once more. If it is again unsuccessful, the placenta must be manually removed.

Manual removal of the placenta must never be lightly undertaken, as death may be the result of carrying infective material to the placental site.

Before removing the placenta the vulval and surrounding areas should be surgically prepared with iodine, the cord appearing outside the vulva should be cut off and clamped, and then the gloved hand should be passed up along the cord to the placenta. The fingers should now be insinuated between the edge of the placenta and the uterus, and the placenta deliberately and carefully separated until it appears free in the uterine cavity. The other hand is placed on the abdomen to steady the fundus. The intrauterine hand now slowly withdraws the placenta, which should peel off the majority of the membranes. When the placenta is brought as far as the vagina, extraction is completed by pulling on the cord, and then the hand is again passed up to the fundus to seek for any placental tissue which may have remained *in situ*.

Rarely, on attempting manual removal, it will be found impossible to find a line of cleavage between the placenta and the uterus. This condition, known as *placenta accreta*, is due to the absence of the *decidua spongiosum*, with penetration of placental elements into the uterine muscle. A true *placenta accreta* can be adequately dealt with only by hysterectomy, as manual removal is always incomplete and almost invariably followed by severe hæmorrhage, while rupture of the uterus and sepsis are frequent.

There is no necessity to administer an intrauterine douche after manual removal unless hæmorrhage is taking place, though some authorities consider it advisable.

Pituitrin, one cubic centimetre, and some ergot preparation, such as "Aseptic ergot", "Ernutin" or "Gynergen", is now given to attain and to maintain efficient contraction and retraction and thus to prevent any further hæmorrhage.

The patient's general condition may be improved by warmth, by raising the foot of the bed and by giving rectal injections of glucose and saline solution.

Post Partum Hæmorrhage.

Primary *post partum* hæmorrhage is abnormal hæmorrhage occurring within twenty-four hours of the birth of the child. It may arise either from the placental site or from lacerations of the genital canal.

Placental site hæmorrhage is due to deficient retraction of the uterine muscle, general causes being debility, grave anæmia and prolonged anæsthesia or sedatization, while local causes are a full bladder, uterine inertia, *placenta prævia*, fibroids and bad management of the third stage of labour.

Traumatic hæmorrhage follows injuries to the soft parts where large blood vessels are implicated. Internal manipulations and forcible extraction of the child are the most frequent causes.

The occurrence of *post partum* hæmorrhage is preventible in the majority of cases.

A definite routine treatment should be adopted. At the onset of hæmorrhage the fundus of the uterus should be massaged to induce it to contract, but if the bleeding then persists, Credé's expression should be done. If expression of the placenta is unsuccessful, it should be removed manually, as described above, and then pituitrin and "Gynergen" should be injected intramuscularly. Should the uterus not become contracted after manual removal of the placenta, a hot intrauterine douche at a temperature of 48·8° C. (120° F.) should be given, if immediately available.

If the hæmorrhage still continues, the uterus should be kneaded and compressed between one hand in the vagina and the other on the abdomen, and as a final resort the uterus and vagina must be swiftly packed with roller gauze. The uterus is then acutely anteverted over the pubes and held in place by an abdominal pad and binder. Firm and thorough packing will stop practically any *post partum* hæmorrhage from the placental site.

Severe and urgent cases must be treated with the utmost promptitude, as any delay may jeopardize the safety of the patient.

Where the bleeding is not gross but a small gush appears each time the uterus contracts, the attendant should not delay, but should express or manually remove the placenta before the patient is allowed to become exsanguinated. It must further be remembered that the greater the loss of blood, the greater the loss of uterine tone, which in turn induces further hæmorrhage, and the greater the risk of infection due to lowered resistance.

With traumatic hæmorrhage the bleeding takes place even when the uterus is firmly contracted. The bleeding point should be searched for and secured. A posterior speculum is useful in bringing into view injuries in the upper part of the vagina, while tears in the cervix may be exposed by downward pressure on the fundus.

Three other rare forms of *post partum* hæmorrhage may be mentioned. Both that from a ruptured uterus causing intraperitoneal hæmorrhage and that from trauma to vessels in the broad ligament causing a pelvic hæmatoma are best treated by abdominal section and, usually, hysterectomy. Acute inversion of the uterus, which may produce severe hæmorrhage, should be immediately replaced, with removal of the placenta.

Secondary *post partum* or puerperal hæmorrhage is that occurring more than twenty-four hours after delivery. It commonly occurs between the fifth and eighth day and is usually due to the retention of placental tissue or, rarely, to a submucous fibroid. Treatment consists in exploring the uterus with the finger and in the removal of any placental remains by curage. If the material cannot be separated, blunt curettage may be necessary, although it is dangerous to use a curette in a puerperal uterus. A hot intrauterine douche should then be administered, and if then there is still bleeding the uterus should be packed.

In regard to restorative treatment, although the first indication in the treatment of *post partum* hæmorrhage is to stop the bleeding, the general condition of the patient also requires prompt attention.

With moderate blood loss this should be replaced by normal saline solution given rectally, subcutaneously or by the intravenous route, and in severe cases blood transfusion should be performed. The patient is placed with the head low and packed with hot water bottles. Elevating and bandaging the arms and legs may be used to increase the amount of blood circulating in the body and head. Stimulants, such as brandy, strychnine or "Coramine", are useful, while small doses of morphine are of value in combating restlessness. During the period of convalescence liberal nourishment, fresh air and iron are of the utmost importance.

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British Medical Association News.

SCIENTIFIC.

A MEETING OF THE TASMANIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held on October 9, 1934, DR. E. BRETTINGHAM MOORE in the chair.

Addison's Disease Treated by "Eschatin".

DR. TERENCE BUTLER reported a case of Addison's disease in which the patient was successfully treated with "Eschatin".

The patient was a tall, thin woman, aged sixty years. She was treated for high blood pressure in November, 1928. The blood pressure readings were then 180 millimetres of mercury systolic and 110 millimetres diastolic. Her weight was 54·9 kilograms (eight stone ten pounds). She suffered from severe constipation. She had been more or less under observation for high blood pressure ever since this date, the average readings being about 180 millimetres of mercury systolic and 110 millimetres diastolic. Very little difference could be effected by treatment. During this period she developed a moderate degree of rheumatoid arthritis; her weight varied between 56·25 and 51·3 kilograms (eight stone thirteen pounds and eight stone two pounds).

On April 21, 1933, the patient had consulted Dr. Butler; she complained of loss of appetite and weight. She stated that she felt weak and ill and suffered from abdominal pain which could not be localized, but which seemed to be situated more in the upper region. Her systolic blood pressure was 156 and her diastolic pressure 100 millimetres of mercury. Her weight was 48·15 kilograms (seven stone nine pounds). On May 5, 1933, she was feeling a little better.

On May 15, 1933, she was about the same, but she was now complaining of thirst and of cramp in the hands resembling tetany. She was given calcium, parathyreoid extract and vitamin *D*.

On May 22, 1933, she was feeling much better. Her weight was 48·26 kilograms (seven stone nine and a quarter pounds).

On June 6, 1933, she was feeling much worse and her weight was 45·6 kilograms (seven stone three and a half pounds). Her systolic blood pressure was 162 and her diastolic pressure 104 millimetres of mercury. She was examined by X rays, but no malignant disease was found. A general atonic condition of the stomach, intestines and large bowel was present, but this had been observed several years before.

On June 10, 1933, she was forced to remain in bed; she was weaker and was feeling very ill; her appetite was very poor, and the skin of her face, neck and hands was considered to be darker than usual. Her systolic blood pressure was 160 and her diastolic pressure 98 millimetres of mercury.

On June 12, 1933, her condition was the same and her systolic blood pressure was 160 and her diastolic pressure 108 millimetres of mercury.

On June 25, 1933, her systolic blood pressure was 158 and her diastolic pressure 104 millimetres of mercury.

On June 26, 1933, her systolic blood pressure suddenly dropped and was 110 millimetres of mercury; her diastolic pressure was 88 millimetres. A diagnosis of Addison's disease was then made. This diagnosis was confirmed at a consultation on June 29.

On June 31, 1933, "Eschatin" injections were commenced, two cubic centimetres a day being given. However, no improvement was noticed and the dose of "Eschatin" was increased in the middle of July to 2·5 cubic centimetres daily with 0·3 cubic centimetre (five minims) of adrenaline; the patient was moved to hospital. A slight improvement was noted at this time in her general condition, although she was much more pigmented, especially on the abdomen. Usually her systolic blood pressure was about 70 and her diastolic pressure 95 millimetres of mercury.

The patient maintained this condition until early in October. During this period an attempt was made to replace some of the "Eschatin" by adrenaline, in order to reduce the expense, which was enormous. This was disastrous, and during one injection of adrenaline, 0·48 cubic centimetre (eight minims), the patient collapsed very badly. After this 0·18 cubic centimetre (three minims) of adrenaline was the maximum dose used, and 3·0 cubic centimetres of "Eschatin" daily.

On October 10, 1933, she was moved to another hospital. Several days before this she had not seemed as well. On the next day she had a very severe exacerbation. The pulse rate dropped to 42 per minute and was very irregular. She vomited most of the food she could be induced to take. The respirations were laboured and she seemed on the point of death. Photophobia was pronounced.

The "Eschatin" was increased to six cubic centimetres per day. More "Eschatin" would have been given, but large supplies could not be procured at short notice.

At a consultation it was seriously debated whether it was worth while continuing the "Eschatin" in view of the expense, and as the patient seemed almost sure to die in any case. However, she was kept on "Eschatin" and she gradually improved.

After a month she was able to read, and this improvement continued until January, 1934, when she was able to get up for the first time. Her systolic blood pressure was usually about 110 and her diastolic pressure 90 millimetres of mercury. The pigmentation was also clearing up in waves about three millimetres (one-eighth of an inch) wide, having a curious appearance like ripples left in the sand by the receding tide.

The patient's appetite was improved and she was getting fatter and sitting outside for a portion of the day; but she still complained of pains in the abdomen. The "Eschatin" dose was 3·0 cubic centimetres per day and the adrenaline 0·18 cubic centimetre (three minims). Previously large doses of salt were tried, given in capsules, but they acted like an emetic and had to be discontinued. Cotton seed oil she refused to take after one dose.

On January 30, 1934, the patient was not so well and was very irritable, and a few days afterwards photophobia developed and she had another exacerbation, associated with loss of appetite, bradycardia, vomiting and air hunger. The pulse rate fell to 45 per minute. To attempt to relieve this and the general depression of spirits, large doses of thyreoid extract were tried (0·6 gramme or ten grains per day). This certainly seemed to improve the pulse rate, but Dr. Butler said that in retrospect he thought that perhaps it was a wrong treatment, as it had been shown that thyreoidectomized animals lived longer than those not so treated when the suprarenals were removed.

After about six weeks the patient's condition improved, but she was unable to get up. The "Eschatin" dosage was maintained at five cubic centimetres per day. In April she had another crisis, again ushered in by irritability and photophobia. The "Eschatin" was increased to six cubic centimetres per day. This attack was, however, not nearly so severe as the previous attacks and no anxiety was felt for her life. By the end of May she was well enough to read again and was obviously putting on weight. In July she started to get up, and by August 24 she left the hospital.

The dose of "Eschatin" had been maintained at 5·0 cubic centimetres with 0·18 cubic centimetre (three minims) of adrenaline and 0·48 gramme (eight grains) of thyreoid extract per day. The pigmentation, which had returned with each exacerbation, was again clearing up in waves, although the abdomen was still very dark—about the colour of a light negro. The thyreoid extract was discontinued and the "Eschatin" was reduced by 0·5 cubic centimetre per week. Salt was again tried in capsules, but it acted again as an emetic. However, she takes large quantities with her food and can retain this quite well.

Dr. Butler said that the patient's present condition was that she could walk a mile or more a day and do her house work and gardening. On October 4 her systolic blood pressure was 142 and her diastolic pressure 100 millimetres of mercury. Her weight was 55·8 kilograms (eight stone twelve pounds). The pigmentations had practically disappeared. She still had some "dyspepsia". The patient was receiving 1·0 cubic centimetre of "Eschatin" and 0·18 cubic centimetre (three minims) of adrenaline a day.

Dr. Butler concluded that it would seem that, no matter how hopeless a case of Addison's disease appeared to be, if one could carry on treatment long enough, the suprarenals had the power of recovery to some extent, as this patient was now quite well upon a dose that one year previously would not have maintained life.

Dr. Butler intended to reduce gradually the maintenance dose still further as long as the patient was well. Fortunately the price of "Eschatin" had been almost halved in the last few months.

DR. F. W. FAY thanked Dr. Butler for his report and congratulated him on the success of his treatment. He said that he had seen the patient in consultation in June, 1933, and that he had thought at that time that the patient was doomed to die in a little while.

DR. S. G. GIBSON also congratulated Dr. Butler and said that the patient was fortunate in being wealthy enough to pay for the "Eschatin", which, he understood, had already cost over eight hundred pounds.

A MEETING OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at Saint Vincent's Hospital, Melbourne, on November 21, 1934. The meeting took the form of a series of clinical demonstrations by members of the honorary staff. Part of the report of this meeting was published in the issue of March 16, 1935.

Hæmoptysis and Diagnosis of its Cause.

DR. W. J. NEWING showed four patients to illustrate difficulties in the diagnosis of hæmoptysis.

The first patient was a healthy looking married woman, aged twenty-six years, whose first indication of sickness was a severe hæmoptysis. Physical examination of the lungs revealed nothing beyond scattered râles, and did not suggest tuberculosis. The heart, however, was very large and of the aortic type; the apex beat was in the sixth intercostal space, 15 centimetres (six inches) from the middle line, with very little cardiac dulness. A soft systolic bruit was audible all over the precordium, with maximum intensity near the apex. X ray examination demonstrated a diffuse mottling of both lungs, which

necessitated the diagnosis of tuberculosis being excluded. The cardiac shadow was that of great ventricular enlargement, with a very small aorta and no demonstrable aortic knob. The arteries in the chest wall were not prominent, but there were some small areas of decalcification of the ribs in the region of the perforating arteries. There was no cyanosis and very little dyspnœa on exertion. The patient's systolic blood pressure was 125 and the diastolic pressure 80 millimetres of mercury.

Dr. Newing thought the condition was probably one of coarctation of the aorta, the pulmonary shadows being due to pulmonary congestion.

The second patient was a woman, aged twenty years, who, three years previously, had been in hospital with a profuse hæmoptysis. A period of bronchopneumonia followed. Examination at that time revealed a moist condition of the right lower lobe, with numerous coarse râles. After recovery a lipiodol injection was given and demonstrated dilatation of the bronchial tubes in the right lower lobe, the remainder of both lungs being quite clear. The sputum was examined several times and no tubercle bacilli were found. Bronchiectasis was diagnosed and a successful artificial pneumothorax was carried out for two years. The physical condition at this time was excellent.

Six months after the lung was allowed to reexpand the patient's condition began to decline and she developed frank tuberculosis of the right upper lobe.

The third patient was a girl, aged fourteen years, who had been admitted to hospital two years previously with profuse hæmoptysis and a history of having coughed blood periodically since her early years. Bronchopneumonia of the right lower lobe supervened. After recovery physical examination revealed no abnormality. X ray examination revealed a small patch of increased density in the right middle lobe, and lipiodol injection showed this to contain an irregular sinuous cavity, about one centimetre in diameter and three to four centimetres in length. The patient's physical condition was excellent and in between hæmoptyses there were no symptoms. Artificial pneumothorax had now kept the bleeding in check for two years.

Dr. Newing considered the condition to be one of bronchiectasis.

The fourth patient was a woman, aged fifty-six years, who had been admitted to hospital in an exsanguinated condition from hæmoptysis. Smaller bleedings had occurred previously on several occasions; cough and sputum had been present for some years. Physical examination revealed a condition of generalized pulmonary fibrosis with patches of râles scattered throughout both lungs, suggesting a condition of widespread bronchiectasis. X ray examination and lipiodol injection confirmed this suspicion. The sputum did not contain any tubercle bacilli.

NOMINATIONS AND ELECTIONS.

The undermentioned have been elected members of the Victorian Branch of the British Medical Association:

Baldwin, Godfrey Joseph, M.B., B.S., 1932 (Univ. Melbourne), Yallourn Hospital, Yallourn.

Buntine, Robert Douglas, M.B., B.S., 1933 (Univ. Melbourne), 31A, Toorak Road, Malvern.

Catarinich, John, Junior, M.B., B.S., 1932 (Univ. Melbourne), Mental Hospital, Mont Park.

Collins, Vernon Leslie, M.B., B.S., 1933 (Univ. Melbourne), Box 141, Nhill.

Keogh, Esmond Venner, M.B., B.S., 1927 (Univ. Melbourne), Commonwealth Serum Laboratories, Parkville, N.2.

Holdsworth, William Goldthorp, M.B., B.S., 1933 (Univ. Melbourne), Baptist College, North Melbourne.

Dowling, Philip Gerard, M.B., B.S., 1932 (Univ. Melbourne), Eye and Ear Hospital, East Melbourne.

Medical Societies.

VICTORIAN MEDICAL WOMEN'S SOCIETY.

The annual meeting of the Victorian Medical Women's Society was held at the Lyceum Club on the evening of Monday, March 11, 1935. Dr. Lucy Bryce presided. There was an attendance of 34 members.

The Secretary reported that eight monthly meetings of the Society had been held during 1934. Papers were read by Dr. Janet Grieg, Dr. Helen Shaw, Dr. Jean Littlejohn, Dr. Olive Buckley, Dr. Adelaide Gault, Dr. Mary Herring, and Sister Lelean, dietitian at the Melbourne Hospital. One clinical meeting was held at the Queen Victoria Hospital, at which cases of medical and obstetric interest were shown by Dr. Kate Mackay, Dr. Marian Wanliss and members of the obstetric staff.

Dr. Constance Ellis represented the Society during 1934 on the Council of the Victorian Branch of the British Medical Association and had again been elected to that office in 1935.

Dr. Roberta Donaldson acted as corresponding secretary for the International Federation of Medical Women.

The hope was expressed that some meeting of the Australian Federation of Medical Women might be arranged at the time of the British Medical Association Annual Meeting in September, 1935.

The death of Dr. Lillian Alexander, a founder of the Society and a past office-bearer, was recorded with deep regret. Also in January, 1935, the death occurred after a tragic accident of Dr. Stella Symes.

The office-bearers for 1935 were elected. Dr. Lucy Bryce was reelected as President. Dr. Bruce Scott was appointed Honorary Secretary.

Obituary.

JOHN WILLIAM DUNBAR HOOPER.

Dr. John William Dunbar Hooper, whose death has been recorded in these pages, was one of those followers of the medical profession who worked continually for its advancement and who strove by every means in his power to advance the common good. His services to the medical profession of Victoria and, through this journal, to the wider sphere of medicine in the Commonwealth were of the greatest importance.

Dunbar Hooper was born in October, 1860, at Dinapore, India. He was the eldest son of the late Sir William Roe Hooper, sometime Surgeon-General of the Indian Medical Service and Honorary Surgeon to His Majesty the King, a "Bart's" man, and a friend of Lister. He went to school at Croydon, at Epsom and at Stratford-on-Avon. He studied medicine at the University of Edinburgh and qualified as Licentiate of the Royal College of Physicians and of the Royal College of Surgeons at Edinburgh in 1883. He subsequently took the degree of Doctor of Medicine at the University of Durham. During his student days at the University of Edinburgh he had Milne Murray as his coach for part of the time. His first appointment was at Lossiemouth, and his second at Elgin. He then spent a year in India and subsequently came to Australia.

In his practice Dunbar Hooper devoted considerable attention to gynæcology and obstetrics. He was for many years associated with the Women's Hospital and served for a period as honorary surgeon. Owing to his interest in the obstetrical art he was included in the committee appointed by the Council of the University of Melbourne to advise the University on the best method of carrying out the wishes of the Trustees of the Edward Wilson (*The Argus*) Trust when it gave a sum of £10,000 for the purpose of endowing research in the science of obstetrics. He was

also one of those who, on the publication of the report of the investigations carried out by Dr. R. Marshall Allan with the aid of this endowment, advocated the appointment of a whole-time professor in obstetrics in the University of Melbourne.

The British Medical Association naturally attracted the attention of a man like Hooper. He was elected a member of the Association in 1887. The Victorian Branch owes much to his energy and devotion. From 1917 to 1920 he acted as honorary secretary; his term of office was one of much activity. On his retirement from the office in 1920 the following statement was included in the annual report of the Council:

"Dr. J. W. Dunbar Hooper resigned the position of Honorary Secretary, which he had held for the previous four years. Dr. Hooper's term of office was a strenuous one, embracing almost the whole of the war period and that of the lodge dispute. He rarely missed a meeting of the Council or of the Organization Committee, which for some time met five days a week. Not only his energy, but his unfailing tact in handling delicate situations was of the greatest service to the Council and to the members generally. Only when the lodge dispute was fairly settled did he feel free to resign from the office which had taken up such a large amount of his time at so large a personal sacrifice. The Council placed on record its appreciation of his great services to the Association."

He was President of the Victorian Branch in 1924 and President of the Section of Gynæcology and Obstetrics at the Second Session of the Australasian Medical Congress (British Medical Association), held at Dunedin in 1927. He was one of those who founded the Melbourne Permanent Post-Graduate Committee and was for many years its honorary secretary. Dr. W. W. S. Johnston and Dr. Fay Maclure have, in their appreciation, shown how much Hooper had to do with the present satisfactory position of the committee. Dunbar Hooper also took a keen interest in the work of the Australian Inland Mission; even when he retired from active practice in 1930 he continued to act as a member of its board.

This short summary of some of Hooper's activities will show how well he earned the esteem and goodwill of his brother practitioners. Those who knew him as a friend held him in high regard and affection. He has been described as a loyal, upright and unselfish friend; there could surely be no finer tribute.

Dr. W. W. S. Johnston and Dr. Fay Maclure write:

Dr. Hooper was one of a band of enthusiasts who were instrumental in launching the Melbourne Post-Graduate Committee in 1920. The purpose behind the movement was that of enabling medical men who, through exigencies of active service, had been unable to keep abreast of the latest developments, to do so through the medium of intensive and practical courses of instruction. Amongst eager workers in the cause were such men as the late Dr. A. V. M. Anderson (first chairman of the committee), Dr. S. W. Patterson, and Dr. J. H. Anderson.

Dr Hooper was appointed the first honorary secretary, a position in which his especial qualities of tireless enthusiasm and eagerness to help the younger men of the profession had great scope. His genial and sociable nature had resulted in the formation of many friendships, and his influence was felt in many sections of the community. An instance of this was forthcoming in 1924, when, owing to his efforts, a sum of £200 was presented to the committee for the institution of a special prize on obstetrics, this being awarded for the best essay on "The Causes and Prevention of Maternal Morbidity and Mortality".

As time went on, the duties of the honorary secretary became more onerous, and in 1923 a co-secretary was appointed, Mr. H. R. Dew, now Bosch Professor of Surgery in the University of Sydney, being selected. Dr. Hooper continued to act as one of the joint honorary secretaries until 1929, when, to the profound regret of the committee, he decided to resign. He had been an ideal person for the position; his courtesy was unfailing, his thought for all who helped the committee in however humble a way, was unbounded, and innumerable kindly actions performed quite unobtrusively helped on the work of the committee, while in a broader field his hospitality to visitors still remains as a charming memory to many from overseas, and serves to keep contact between workers here and in other parts of the world.

Until his resignation from the Council of the Victorian Branch of the British Medical Association in 1930 Dr. Hooper had, from its inception, acted as one of the representatives of the British Medical Association on the committee, and although he suggested retiring, he was induced to accept cooption, and in this capacity remained as a member of the committee until his final resignation in 1932. He retained his interest in the affairs of the committee, and as recently as September last invited Professor David Barr, a visiting lecturer from St. Louis, United States of America, to a pleasant little gathering at his home. It was his last act performed for the committee, and a fitting one to close with, revealing in characteristic fashion his kindliness and his feeling of friendship for the traveller from overseas.

JOHN FRANCIS WILKINSON.

We regret to announce the death of Dr. John Francis Wilkinson, which occurred at Melbourne, Victoria, on March 29, 1935.

Correspondence.

"DETTOL."

Sir: Dr. Constance D'Arcy, in the Anne MacKenzie Oration, stresses the value of "Dettol" as an antiseptic in midwifery. This I can wholeheartedly subscribe to from my experience as resident obstetrical surgeon at Saint Mary's Hospital, Manchester, 1933-1934. Over a period of months I used "Dettol" only as an antiseptic in the "urgency ward" (that is, cases having interference before arrival, failed forceps, delayed labour *et cetera*) against usual antiseptics in the other wards. "Dettol" came through with marked success, particularly so because of its portability and usefulness. A single bottle of "Dettol" in the bag supplies a multiplicity of purposes. Used pure, "Dettol" (undiluted) on hands and skin is efficient and non-injurious; used diluted for swabs, instruments *et cetera* it is equally effective. If objected to in "milky" solution for instruments, it is also obtainable in special colourless type.

At present I am experiencing extreme difficulty in obtaining supplies in Australia and would be grateful for the name of the agent.

Yours, etc.,

W. J. Rawlings, M.B., B.S., M.C.O.G., D.G.O. (Dublin).

Corowa,
New South Wales,
March 5, 1935.

ALLERGY AND PNEUMONIA.

Sir: Your subleader in the March 23 issue of your journal, on "Allergy and Pneumonia", is of much interest, as it helps to confirm views which I have expressed in the January 12 issue, in "The Relationship of Allergy to the Autonomic Nervous System". It has long been a puzzle why individuals carrying a virulent infection for years escaped serious damage, then all of a sudden seemed to succumb to some arthritic, kidney, cardiac or other condition. It has been regarded by many that for some time the defence forces were able to hold out, then eventually failed. Exactly how they failed was not clear, but the results of these experiments open up a new solution to the question. Carrying an infection in one's tonsils, sinuses, or around one's teeth necessarily means a certain amount of blood invasion, in other words, a daily injection of so many streptococci, which must sooner or later produce what is known as the arthus phenomena in some particular tissue. Why certain tissues are picked out to the exclusion of others is probably due, as Rosenow points out, to the affinity of that particular strain of organism for that tissue, but the fact remains that that tissue has become sensitized, or the organisms or their toxins would not have been held up there. So with the presence of the organisms there is a condition of inflammation *plus* allergy, whilst with the presence of toxin alone the reaction is purely allergic. Pneumococci are frequently associated with infected sinuses, so there is the possibility of any so infected to succumb to pneumonia when the environment is suitable.

The exudation of the pneumonic lung is somewhat akin to that in an urticarial wheal, but in the former it is probably a condition of allergy *plus* inflammation, whilst in the latter, pure allergy. As adrenaline is specific in the latter, it may be of some use in the former, should the allergic phenomena be dominant. These facts should make us bear in mind the possibility of any tissue being liable to allergic exudations, especially with reference to epilepsy, and also the enormous field that the sympathetic and parasympathetic systems, with their accompanying internal secreting glands, play in disease.

Yours, etc.,

Sydney Pern.

12, Collins Street,
Melbourne,
Undated.

Post-Graduate Work.

LECTURES IN ADELAIDE.

Owing to the annual meeting of the British Medical Association being held in Melbourne from September 9 to 14 next, a post-graduate course will not be held in May, 1935. The Adelaide Permanent Post-Graduate Committee has arranged, however, for a series of twelve lectures (six medical and six surgical) to be delivered on Thursday evenings, at 8.30 o'clock, in the Anatomy Lecture Theatre, University of Adelaide, commencing May 2, 1935.

The lectures will be on conditions commonly met with in general practice and will deal with the practical side of the subjects and, where possible, they will be in the nature of lecture demonstrations.

Duplicated copies of the lectures will be provided for those attending the course, either in full or condensed form, and copies for country practitioners will be available at a cost of £2 2s. per set.

The programme is as follows:

May 2: "Treatment of Pulmonary Tuberculosis", Dr. D. R. W. Cowan.

May 9: "Pre-Operative Treatment: Preparation of the Normal and Abnormal Case for Operation", Dr. I. B. Jose.

May 16: "Diabetes Mellitus: Some Remarks on Modern Methods of Treatment, including Coma, and Preparation for Operation", Dr. A. R. Southwood.

May 23: "Treatment and Diagnosis of the Cause of Acute Retention of the Urine", Dr. G. H. Burnell.

May 28: E. C. Stirling Lecture: Dr. Victor Hurley, of Melbourne.

May 29: E. C. Stirling Lecture: Dr. Victor Hurley, of Melbourne.

June 6: "Cardiac Irregularities: Their Diagnosis and Treatment", Dr. W. Ray.

June 13: "Treatment of Fractures of the Clavicle and Upper End of Humerus", Dr. A. F. Hobbs.

June 20: "Pathology and Diagnosis of Tumours in Region of Sella Turcica", Dr. C. T. de Crespigny.

July 4: "Treatment of Wounds", Sir Henry Newland.

July 11: "Modern Views on Causation and Treatment of Rheumatoid Arthritis", Dr. Frank S. Hone.

July 18: "The Diagnosis of Tumours of the Breast", Dr. H. R. Pomroy.

August 1: "Pathology, Diagnosis and Treatment of Pyelitis in Children", Dr. R. L. Thorold Grant.

August 8: "The Treatment of the Abnormal Conditions of the Feet", Dr. P. S. Messent.

The following are the fees for the lectures: Full course of twelve lectures (excluding E. C. Stirling Lectures), £4 4s.; course of six lectures (excluding E. C. Stirling Lectures), £2 2s.; single lecture, 10s. 6d.; E. C. Stirling Lectures, £1 1s.

Those desiring to attend should notify the Lay Secretary of the Branch (Mr. Walter C. Dobbie) either by telephone (Central 4256) or letter as soon as possible. Early application for tickets is especially requested; this will greatly facilitate the necessary arrangements.

Books Received.

RADIOLOGICAL TERMINOLOGY, by C. E. Gaitskell, M.R.C.S., L.R.C.P.; 1935. London: J. and A. Churchill. Foolscap 8vo., pp. 90. Price: 5s. net.

THE PHARMACOLOGICAL ACTION OF THE HARROGATE DRINKING WATERS, by W. Bain, M.D., F.R.C.P., F.R.C.S.; 1935. London: J. and A. Churchill. Demy 8vo., pp. 60, with 26 illustrations. Price: 5s. net.

STATISTICAL METHODS FOR RESEARCH WORKERS, by R. A. Fisher, Sc.D., F.R.S.; Fifth Edition, revised and enlarged; 1934. Edinburgh: Oliver and Boyd. Demy 8vo., pp. 332. Price: 15s. net.

THE WORLD OF MAN AS REFLECTED IN ART, IN WORDS AND IN DISEASE, by G. Groddeck, M.D.; 1934. London: The C. W. Daniel Company. Crown 8vo., pp. 271, with illustrations. Price: 10s. 6d. net.

Diary for the Month.

Apr. 16.—New South Wales Branch, B.M.A.: Ethics Committee.
Apr. 16.—Tasmanian Branch, B.M.A.: Council.
Apr. 17.—Western Australian Branch, B.M.A.: Branch.
Apr. 23.—New South Wales Branch, B.M.A.: Medical Politics Committee.
Apr. 24.—Victorian Branch, B.M.A.: Council.
Apr. 24.—New South Wales Branch, B.M.A.: Branch.
Apr. 25.—South Australian Branch, B.M.A.: Branch.
Apr. 26.—Queensland Branch, B.M.A.: Council.
May 1.—Western Australian Branch, B.M.A.: Council.
May 2.—South Australian Branch, B.M.A.: Council.
May 3.—Queensland Branch, B.M.A.: Branch.
May 6.—New South Wales Branch, B.M.A.: Organization and Science Committee.
May 7.—Tasmanian Branch, B.M.A.: Council.
May 10.—Queensland Branch, B.M.A.: Council.

Medical Appointments.

Dr. D. F. Buckle has been appointed Medical Officer, Department of Mental Hospitals, New South Wales.

* * *

Dr. K. F. Abernethy (B.M.A.) has been appointed Medical Officer of Health by the Kojonup District Road Board, Western Australia.

* * *

Dr. H. H. Bullmore (B.M.A.) has been appointed Honorary Physician at the Prince Henry Hospital, Little Bay, New South Wales.

* * *

Dr. P. Parkinson (B.M.A.) has been appointed Honorary Radio-Therapeutist at the Prince Henry Hospital, Little Bay, New South Wales.

* * *

Dr. K. O. Jones (B.M.A.) has been appointed Government Medical Officer at Campbelltown, New South Wales.

* * *

Dr. L. W. Linn (B.M.A.) has been appointed Honorary Dermatologist at the Parkside Mental Hospital, South Australia.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xv, xvi, xvii.

Blair Athol District Hospital, Blair Athol, Queensland: Medical Officer.
Children's Hospital (Incorporated), Perth, Western Australia: Junior Resident Medical Officer.
Maryborough Hospitals Board, Maryborough, Queensland: Junior Resident Medical Officer.
Royal Australian Air Force: Medical Officer.
Sydney Hospital, Sydney, New South Wales: Temporary Honorary Relieving Assistant Physician.

Medical Appointments: Important Notice.

Medical practitioners are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
New South Wales: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
Victorian: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
Queensland: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associate Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointment and those desiring to accept appointments to any COUNTRY HOSPITAL, are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
South Australian: Secretary, 207, North Terrace, Adelaide.	Officer of Health, District Council of Elliston. All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
Western Australian: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
New Zealand (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

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